THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—20TH YEAR. SYDNEY, SATURDAY, APRIL 29, 1933. No. 17.

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COAL MINER'S LUNG: A PRELIMINARY ACCOUNT OF THE CHEMICAL ANALYSIS AND PATHOLOGY OF THE LUNGS OF COAL MINERS IN NEW SOUTH WALES.[1]

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THIS investigation deals with the chemical analysis and pathology of the lungs of thirty-one persons. Of these nineteen worked as coal miners, six had done more or less metalliferous mining, three were Sydney sandstone masons, three were metalliferous miners, two were engaged in other dusty occupations (a metal polisher and an ore miller), two were not exposed to dust in their calling, and two had died from tuberculosis and had no known dust exposure. This investigation is a continuation of previous work.

Up to the present isolated and incomplete analyses have been published by others of the free silica in the lungs of men who had pulmonary fibrosis caused by dust, and in the absence of a description of the chemical method used these are of little help.

In the group analyses which have so far appeared, the chief of which are those of McCrae (1913) and of Cummins and Sladden (1930), the total silica was determined and this was not separated into free silica or quartz and combined silica or silicates.

McCrae dealt with lungs of South African silicotics, and it could safely be assumed that much of the total silica returned was quartz, but with the coal miners of Wales, whose lungs were analysed

[1] This paper is Number 17 of "Studies in Industrial Hygiene" from the office of the Director-General of Public Health, New South Wales.

by Cummins and Sladden, no such assumption could be made. We should have preferred to delay publication of our results until our series of lungs was larger and further pathological work had been done, but we owe a duty to those medical practitioners who have often, at considerable inconvenience to themselves, aided us in this matter, and who desire to see a preliminary comparative report on the lungs we have examined, that they may appreciate individual reports on lungs submitted by them.

Moreover, the results of this investigation are so interesting that they justify a preliminary account. The characters of the Welsh coal miner's lung portrayed by Cummins and Sladden are found in our affected coal miners: the upper lobe consolidation, with non-tuberculous cavitation, appears and the prolongation of this primary consolidation to form a cuirass over the exterior of the upper parts of the lungs is seen. An important result comes from the analysis of the lungs of a collier who worked for thirty-two years in the same pit, a South Coast colliery.

The coal dust to which this man was exposed for his working life contained only 0·4% of free silica. His lungs had upper lobe consolidations with marked generalized nodular fibrosis, yet they contained no more free silica than those of a normal person, and the lungs of an unaffected worker from the same pit showed no increase in free silica.

Here, then, is a case of gross nodular pulmonary fibrosis not due to free silica; it is a case of silicatosis or an anthracosis, not a case of silicosis or silico-anthracosis (Cummins).

The third interesting result is the discovery that the lungs of a man who worked with a pneumatic tool cleaning out moulding sand in a steel annealing pot, showed massive fibrosis with non-tuberculous cavitation in both lungs. The only similar case we are aware of is the one described by Stern (1929) in a man doing the same class of work in a German foundry.

One suspects that a new problem is nascent and that foundry worker's lung will present features allied to coal miner's lung. Our last two cases show that knowledge of pulmonary dust fibrosis is still limited and awaken interest in ideas of causation, such as the varied stimulation of dust cell activity by different dusts. It is the anthracite coal in Wales that produces in that country most fibrosis, and in New South Wales it is a coal of low hydro-carbon content as compared with our other coals.

Reference might be made here to the tenacity with which carbon retains silica and silicates, for with caustic soda it is very difficult to extract silica from coal dust, and it is probable that there exists the same insolubility of silica *plus* carbon in body fluids. It is hoped, from investigations which are proceeding, that it will be possible in analysis of the lungs to differentiate between silica and silicates of inhaled coal dust and these minerals derived from other sources, using this fact that the silica and silicates originally associated with coal are not acted upon as easily as when they are inhaled from minerals other than coal. Coal dust of special character (apart from the silica, free and combined, which it may contain) in heavy inhalations, may yet prove to be a potent cause of fibrosis.

We are indebted to Mr. H. F. Whitworth, of the Department of Mines, for the following:

I have compared Helensburgh coal with others from the South Coast, and also from the Newcastle, Maitland (Northern) and Lithgow (Western) fields, and find from the fixed carbon volatile ratio that it has closer affinities to the anthracites than any other New South Wales coal, with the possible exception of coal from the Sydney Harbour Colliery. This point probably has some significance, as your lung analyses show a high carbon content for Helensburgh miners.

I have procured typical coal ashes from Greta and Newcastle coals for comparison with the Helensburgh coal ash, and find in each case that they are more clayey and contain less definite mineral matter than the Helensburgh coal ash. The latter contains a considerable percentage of felspar fragments and also an unknown mineral occurring in tiny prismatic crystals which has been observed in lung ash. In view of Dr. Taylor's analysis of Helensburgh ash, showing an abnormally high percentage of alumina, I suggest that the mineral is one of the high alumina-bearing silicates, such as sillimanite, which is fairly common, and which crystallizes in tiny prismatic crystals similar to those present in the coal ash and lung dusts. In view of the extreme resistance which these high alumina minerals offer to chemical solution, it is possible that they may be a contributing factor in cases of fibrosis amongst workers in the Helensburgh Colliery.

Dust in New South Wales Collieries.

There are three large coal fields within one hundred miles of Sydney: northern, South Coast, and western.

On the northern fields the coal ash averages about 10%; the seams are large and the free silica in the coal is below 1%, except in two mines, in which it approaches 2%. An average figure is 0·6%. The roof, floor, and occasional bands are of conglomerate rock, sandstone, shale, or mudstone, and the free silica averages 26%.

Dust sampling by Greenburg-Smith impinger methods according to our routine practice has in other hands shown an average figure of twenty milligrammes per cubic metre with, in a few samples analysed, no increase of free silica above that found in the coal itself.

In the western pits the ash averages 14%, the free silica is limited and the dust exposure is not undue.

In the pits of the South Coast the coal ash is about 12% with the free silica about 0·5%; the dust in the air is often more marked than in the northern pits, and in one colliery, with pillar extraction, the dust generated is excessive.

Anthracosis in Wales.

Cummins and Sladden, after a prolonged investigation on pathological and histological findings in the lungs of coal miners, supplemented by chemical analyses carried out at Sheffield by Wheeler and Tideswell on the same material, published (1930) an account of 38 cases and concluded that "anthracosis of the lungs is determined by a combination of silicotic fibrosis and accumulation of coal dust" and that "coal miners especially exposed

to silica dust are liable to develop a silicosis which, complicated by the effects of anthracotic dust accumulation, may lead to disablement and death" (Cummins, 1931).

We assume that anthracotic dust accumulation is a dust accumulation producing anthracosis.

These authors describe the pathology of the anthracotic lung: the general blackening—the black india rubber-like consolidation at the apex of the upper or lower lobes and its prolongation downward as a cuirass-like sheath—the fibrotic nodules, the thickened pleura, bullæ and other emphysematous lung changes with non-tuberculous cavitation of the consolidated areas as found in six cases. Cummins (1931) tells how he and Weatherall proved that coal dust can absorb and inactivate tuberculin, and from this explains the relative freedom of coal miners from tuberculosis. He writes:

It must not be supposed, however, that the presence of accumulated coal dust in the lungs is an unmixed blessing. On the contrary it may have very disagreeable or even dangerous results. While it seems to have a definitely beneficial effect in lessening the liability to the extension of tuberculous lesions, it may, by its mere accumulation, come to occupy large areas of lung tissue to an extent which leads not only to fibrosis and devascularization, but sometimes to colliquative changes and even cavity formation. With these changes there is brought about, to a gradually increasing extent, a state of lung emphysema accompanied by structural deterioration of alveolar tissue, bronchioles and even bronchi, which involves serious interference with the function of respiration.

Most old coal miners are dyspnœic. Although many of them work on to a great age, they are, as a rule, "short of breath", and suffer from copious sputum, cough, and sometimes asthmatic trouble.

These authors described lungs from miners of South Wales, who mostly work in anthracite mines and some of them also in hard headings where they are exposed to free silica dust. The records of such exposure of these miners to free silica dust are often indefinite and sometimes absent, and we suggest that analysis to determine the free silica in such lungs might reveal cases where, as in our Case XXII, the fibrosis has resulted from dusts other than free silica. Some of the lungs, however, are akin to those of our coal miners who had metalliferous mining experience with definite exposure to free silica and in whose lungs we found an increase of free silica.

In the account given by Cummins and Sladden of the coal miner's lung two things call for comment. One of these is the loose use of the term silica in the text and conclusions of their article, and the other thing is their belief that petrological examination of lung sections enables them to identify "silica" particles. It will be clear only to keen students of the chemical analysis of lungs that the use of the term "free silica" (on page 1118) is subject to correction, and "total silica" should be read, and we owe thanks to the authors for letters on this matter. We mention this point as it makes a difficulty for many whose attention will be focused on their paper, which is the most recent authoritative account of coal miner's lung. We do not think that free silica or quartz has been shown to exist in these lungs by chemical analysis or by acceptable petrological means; the term silica in the text and conclusions may refer to quartz or silica in combination.

In a later discussion Cummins states (1932):

As to whether the fibrosis resulted only from free silica, or might be due also to silicates, he was unable to say, as the chemist had not yet devised an absolutely reliable method of differentiating the free from the combined silica, either in a sample of dust or a sample of lung; but he did not think that the point was very important either in relation to prevention or treatment.

But to an industrial hygienist it is surely very important to know the chemical nature of the dusts that may produce fibrosis, and in our State and since we are working under absurdly anomalous compensation laws (and this anomaly is not unknown in the Motherland) it is of medico-legal importance and much more is destined to be heard of this point.

Again the question will arise with us as to whether metalliferous miners who become colliers will be more liable to develop anthracosis than men who have worked only in coal mines, and our present outlook suggests that they will; but there is this saving grace, that tuberculosis may not take its toll of them.

One may quote from Haldane's contribution to the discussion:

Almost the only point in which he did not agree with Professor Lyle Cummins was in extending the term silicosis to cover any form of fibrosis due to the inhalation of dust. This seemed inadvisable since there was no evidence that various forms of actual fibrosis were in fact due to inhalation of free silica and not to other forms of dust, such as coal dust or shale dust, which produced no tendency towards tubercular phthisis.

Commenting on the use of the petrological microscope to determine the nature of particles in the lungs of coal miners, we recall that we have been unable in past years, after considerable trial, to use this means in our work on dust sampling in which we desired to enumerate separately quartz and silicate particles up to three microns in diameter.

We have been advised by Mr. H. F. Whitworth, of the Department of Mines, that the usual petrological methods for the determination of minerals are unsatisfactory for the detection of quartz particles in lung sections, as, owing to its low birefringence, quartz does not show interference colours between crossed nicols unless the particles are greater than three microns in thickness. As the average size of the particles of dust in lungs is less than three microns, it is evident that any particles which do show interference colours between crossed nicols must possess a much higher double refraction than quartz. Minerals such as calcite, rutile, zircon, sillimanite and micas will show interference colours even in particles of one micron diameter.

The following statements of Cummins and Sladden will fail to convince those who have conducted such petrological studies that they were examining quartz particles:

There seems little doubt, however, that silica accounts for the majority of the bright points seen with the crossed prisms and, with a little practice, it is possible to distinguish between the minute, pure white and extremely

brilliant light points caused by silica particles and the coarser and often slightly coloured sheen associated with other crystalline substances.

The bright particles were found as a rule to be most numerous in the dust accumulations around the nodules and less numerous in the nodules themselves . . . ."

Cummins (1931) writes in a later article:

Is this condition to be correctly described as silicosis? . . . . Another query as to the accuracy of this definition has been put forward in a private letter addressed to me by Dr. Badham, the Medical Officer of Industrial Hygiene of New South Wales. He asks whether this relatively benign type of lung fibrosis may not be an example of what he has described as "silicatosis" rather than of "silicosis". To this question it is difficult to give an entirely final answer, owing to the difficulty which chemists encounter in arriving at an accurate estimate of the amount of free silica in any given dust. The facts, however, are, in my opinion, all against the "silicatosis" and in favour of the "silicosis" theory as applied to coal miners in South Wales. The petrological characters of some of the hard stone in our coal deposits, and their microscopic characters of the dust in lung sections as seen by polarised light, combine to place the matter as far beyond a doubt as is possible until our chemical analyses can be made more conclusive.

Heard (1932) examined a large number of thin sections of lungs which were in an advanced stage of silicotic fibrosis, and stated:

There appeared to be no direct mineralogical evidence that inhaled quartz fragments occurred in association with the fibrotic nodules. Apart from the statistics derived from cases of miners' silicosis in South Africa, there did not seem to be any conclusive evidence to support the fact that the silicotic fibrosis of British miners was due solely to the inhaling of quartz dust.

He was in complete agreement with Melville Hiley, that "the combined silicates might not be the harmless and innocuous substances they were supposed to be".

Sladden (1932) states:

A large number of chemical analyses had shown beyond any question that silica, either free or combined, was in fact present in entirely abnormal amounts in all these "silicotic" lungs, and it was highly desirable from the pathological standpoint that methods of demonstrating the silica microscopically should be devised.

S. W. Fisher, His Majesty's Medical Inspector of Mines, presenting notes taken at inquests on miners whose deaths had been due directly or indirectly to the inhalation of dusts in mines, says:

It is interesting to note the following points: (i) the frequency of heart dilatation and failure, (ii) the number of non-tubercular cases, (iii) the formation of smooth-lined cavities, (iv) the frequency with which the upper lobes of the lungs are affected, (v) the presence of emphysema and cases of death following the rupture of an emphysematous area.

Radiography.

In Australia the practice of radiography of pulmonary fibrosis has lacked correction in the *post mortem* room and has pursued a course untrammelled by pathology and biological tests of lungs of individuals dead of dust diseases; and this has led to incorrect radiographic interpretation, which has been most marked in skiagrams of coal miners' lungs. The revision of our radiographic standards of dust fibrosis and tuberculosis therewith is called for.

No radiographer is competent to report on an X ray film showing pneumonokoniosis without knowing the man's working history and the nature of the dusts to which he has been exposed. A similar opinion comes from Haldane (*loco citato*). We doubt also whether any radiographer is competent to diagnose tuberculosis in a lung, the seat of dust fibrosis. At his best he can but report the presence of consolidations or unilateral distribution of fibrosis. If his patient has been exposed to free silica, there is something less than an even chance that he will be right in diagnosing tuberculosis when there is consolidation, apical or otherwise. There is something like a ten to one chance that he will be wrong if he reports tuberculosis when there is consolidation, apical or otherwise, and his patient has been a coal miner. Two common factors produce consolidations in lungs—tuberculosis and dust—and it is the clinician's job to determine the presence of tuberculosis by finding tubercle bacilli in the sputum or by noting the presence of an active deteriorating toxæmia along with clinical signs of disease; it is the radiographer's job to report (subject to a correct industrial history) pulmonary dust fibrosis of different types with or without consolidations.

Discussion.

We have been asked to write an account of our present outlook on fibrosis of the lungs in coal miners, particularly from the point of view of causation and our views as to when compensation might, in our opinion, reasonably be claimed.

It will be understood that at present this subject has not been put on a sound pathological, chemical and clinical footing, and only continued study of this condition will make a basis for apportioning the blame for the causation of this fibrosis which will receive the support of most medical men.

After reviewing the chemistry and pathology of these lungs along with the known industrial history and clinical and radiographic findings, we are writing the following.

We appear to have in New South Wales several different types of pulmonary fibrosis in coal miners. One type occurs in coal miners with a history of metalliferous mining before entering the pits, or again there may be no history of exposure to free silica apart from coal mining. Yet another type of fibrosis occurs in our South Coast coal miners, and chiefly in one pit; and the free silica or quartz found in the lungs of two coal miners (one with nodular fibrosis) who worked there is not greater than that found in lungs of normal individuals, but the combined silica (silicates) and carbon were greatly increased. This type of fibrosis may be a silicatosis, a term introduced by one of us (C.B.) to describe fibrosis due to silicates in which the silica exists combined with bases and not as quartz; or it may be a pure anthracosis, that is, a fibrosis due to the carbon of coal dust.

There are, then, in our coal miners several forms of fibrosis. One type is due to exposure to quartz

followed by aggravation due to silicates and other things in coal dust. Another type appears to have resulted from coal dust containing quartz. A third type is due solely to the carbon and silicates of the coal dust concerned and not to a minute amount of free silica or quartz this coal dust may contain.

Chemical analysis and the pathology of the lungs support these views. The question will probably be raised from our work whether it is possible by the chemical analysis to show that there has been an accumulation of coal dust in the lungs apart from any dust which might reasonably come from metalliferous mining. We expect to be able to determine this by chemical analysis by a method already indicated. Such an accumulation of coal dust should, one might reasonably expect, be evidenced by the presence of carbon and combined silica in an amount above that found in the lungs of miners not exposed to coal dust or silicates. When it is necessary to determine during the life of an individual whether he has inhaled sufficient coal dust to aggravate a fibrosis originally due to metalliferous mining, we think it would be reasonable to require an exposure of some years in a pit not particularly dusty, and a shorter time in a dusty pit before one could accept an aggravation or an incitement of a fibrosis due to an earlier inhalation of quartz.

On the evidence at present existing, it is doubtful whether tuberculosis can be aggravated or caused by a coal dust which has a high carbonaceous content.

We consider that the pulmonary fibrosis of coal miners probably renders them more vulnerable to respiratory diseases such as bronchitis and pneumonia. When the fibrosis is advanced and consolidations are present, we consider that cardiac disease with circulatory failure is aggravated.

The basis for compensation should be disability, and as disability may be absent in individuals with well marked radiographic fibrosis, it can be determined only by clinical methods.

The Determination of Silica and Silicates in Lung Tissue.

In the literature dealing with the silica content of lung tissue, no differentiation appears to have been made between silica present as such and silica present in the form of silicates. In an investigation into the effects of these two substances on lung tissue such a differentiation is essential, and it is claimed that the method of analysis which is given in full detail below does enable a differentiation between silica and silicates to be made with a high degree of accuracy.

In the early stages of this investigation attempts were made to determine the silica content of the lung tissue without preliminary ashing. The method employed was to subject the dried lung to the action of sulphuric and nitric acids until all the organic matter was destroyed. Although this method was tedious and cumbersome, a residue was finally obtained which consisted of silica present as such in the lung, silica which had been liberated from silicates by the action of sulphuric acid, and a small quantity of refractive silicates. Owing to the difficulties of manipulation, the amount of dry lung which could be treated in this manner was necessarily small, and consequently the amount of insoluble residue obtained was not large enough to permit the determination of its constituents with any degree of accuracy. This method therefore was abandoned, although at first sight it appeared as though it might have had some advantages over an ashing method on account of the possibility of changes taking place during the period of ashing.

A method has been put forward by King (*Journal of Biological Chemistry*, 1928, Volume XXX, page 25) in which a small amount of dry tissue was treated with nitric acid and ammonium nitrate, the residue was extracted with sodium hydrate, and the silica was finally determined by means of the colour produced with ammonium molybdate.

It is obvious that this method can determine only that silica which is in a form soluble in sodium hydrate. Any silica originally present as such or silicates undecomposed by nitric acid would remain undetermined.

Method of Analysis.

The method of analysis finally adopted was based on the method for the analysis of clays described in Fresenius's "Chemical Analysis", Volume II, and is as follows:

The sample of lung (two hundred to three hundred grammes) is cut into small pieces and dried in a hot air oven at 100° C. until of constant weight. The dry lung is then reduced to a powder in a mincing machine and weighed, and a portion (thirty to forty grammes) is placed in a platinum dish and heated until coked. The coked mass is then ground to a powder and again heated at a temperature which is sufficient to maintain the bottom of the dish at dull redness. The grinding is repeated as required until the ash is perfectly free of carbon. The time required is usually about nine hours.

The analysis of the ash is carried out as follows:

(1) Take 0·5 gramme of ash and place in a small beaker containing five cubic centimetres of pure concentrated sulphuric acid and ten cubic centimetres of distilled water.

(2) Heat gently on a sand bath to strong fuming.

(3) Cool, dilute with fifty cubic centimetres of water and swing in a centrifuge until the supernatant liquid is perfectly clear.

(4) Decant the liquid and transfer the residue from the centrifuge tube to a small beaker by washing out with small amounts of concentrated hydrochloric acid; add a small amount of concentrated nitric acid and heat on a sand bath until five cubic centimetres remain.

(5) Dilute to fifty cubic centimetres with water, centrifuge, decant and wash twice with fifty cubic centimetres of one in ten hydrochloric acid by shaking and then centrifuging (Residue *A*).

(6) Wash the residue into a small platinum dish with thirty cubic centimetres of a 10% solution of sodium carbonate and boil gently for five minutes.

(7) Transfer to a centrifuge tube and swing until the liquid is perfectly clear.

(8) Decant the liquid and determine the silica content as given below.

(9) Wash the residue twice with one in ten hydrochloric acid as in (5).

(10) Transfer the residue to a weighed platinum dish, evaporate to dryness, heat strongly over a flame, and weigh (Residue *B*).

(11) To the weighed residue add two cubic centimetres of hydrofluoric acid, 0·5 cubic centimetre of pure concentrated sulphuric acid, mix well.

(12) Heat on the water bath until most of the hydrofluoric acid has been evaporated, drive off the sulphuric acid, heat strongly, and weigh (Residue *C*).

(13) Add fifty cubic centimetres of water to the residue and heat on the water bath for two hours.

(14) Filter off the undissolved residue, evaporate the filtrate to dryness, heat residue lightly over a flame, and weigh (Residue *D*).

In the above analysis the lung has been treated with sulphuric and subsequently with a mixture of nitro-hydrochloric acid. This was found to be necessary because in some cases when the ash was treated with nitro-hydrochloric acid only, the insoluble residue contained a considerable amount of phosphates which could be decomposed only with concentrated sulphuric acid.

The compositions of the residues obtained from the various stages of the analysis are as follows:

Residue A (Stage 5).—Silica from silicates decomposed by acids, *plus* silica present as such in the lung ash, *plus* undecomposed silicates.

Residue B (Stage 10).—Silica present as such in the lung ash *plus* undecomposed silicates.

Residue C (Stage 12).—Alumina and alkali sulphate.

Residue D (Stage 14).—Alkali sulphate (K_2SO_4). This residue is in most cases very small and has been found to consist almost invariably of potassium sulphate.

By using a method of fractional decomposition it was found that the greater portion of the alumina in Residue *C* was probably derived from a silicate of the form Al_2O_3, $2SiO_2$. The remaining alumina and potassium sulphate are considered to be from a silicate of the form K_2O, Al_2O_3, $6SiO_2$.

The amount of free silica in the ash is given by subtracting from Residue *B* the weight of the undecomposed silicates which can be calculated from Residue *C* by allotting to the K_2SO_4 its correct proportion of Al_2O_3 and SiO_2 according to the formula K_2O, Al_2O_3, $6SiO_2$, and to the residual alumina its correct proportion of SiO_2 according to the formula Al_2O_3, $2SiO_2$.

The amount of combined silica is the sum of the SiO_2 extracted by sodium carbonate (stage 6 of the analysis) and the amount in the two silicates K_2O, Al_2O_3, $6SiO_2$ and Al_2O_3, $2SiO_2$.

The weight of Residue *C* is rarely greater than ten milligrammes, and of Residue *D* very much smaller. For practical purposes Residue *D* may be neglected in the great majority of cases, and the whole of Residue *C* may be taken as Al_2O_3. The undecomposed silicates in Residue *B* then become of the form Al_2O_3, $2SiO_2$.

The determination of the silica extracted by 10% sodium carbonate (stage 6 of the analysis) is made as follows:

Make the decanted liquid up to a volume of 50 cubic centimetres. Take one to five cubic centimetres and place in a 50 cubic centimetre Nessler tube, add 30 cubic centimetres of water and two cubic centimetres of a 10% solution of ammonium molybdate, mix, and add sufficient one in three sulphuric acid to make the solution acid to methyl orange. Make up to 50 cubic centimetres, allow to stand five minutes and then compare the colour produced with the colour of a standard solution of picric acid (0·1024 gramme picric acid in 100 cubic centimetres of water. One cubic centimetre of this solution made up to 100 cubic centimetres represents the colour which is produced by one milligramme of SiO_2 in 50 cubic centimetres of solution.)

For determinations of the constituents of the ash other than silica and the small amount of alumina present in the undecomposed silicates which were carried out on the solutions decanted at stages 4 and 5 of the analysis, the usual methods were employed and it is not necessary therefore to mention them here.

The determination of the coaly matter present in the lung tissue is made as follows:

1. Take two grammes of powdered dry lung and place in a small beaker containing 30 cubic centimetres of 25% caustic soda.
2. Boil gently until all the particles of lung tissue have been dissolved. This usually takes about one hour. The amount of liquid should be maintained at 30 cubic centimetres by the addition of distilled water.
3. Add sufficient concentrated nitric acid to neutralize the caustic soda and then five cubic centimetres in excess.
4. Boil until the liquid becomes brown-yellow in colour; five minutes' boiling is usually sufficient.
5. Transfer the hot solution to a separating funnel, shake vigorously, and allow to stand in an upright position until all the carbon and fat have risen to the top. Run off the liquid as completely as possible.
6. Wash out the beaker with hot alcoholic potash[1] and transfer to the separating funnel, make up to 40 cubic centimetres with hot alcoholic potash, shake vigorously, and transfer to a centrifuge tube; wash out funnel with alcoholic potash.
7. Centrifuge until liquid is free from carbon particles, decant, and wash twice with hot alcoholic potash by shaking and centrifuging.
8. Wash residue twice (as in 7) with acid alcohol.[2]
9. Transfer the residue to a weighed platinum dish by means of small amounts of water, evaporate to dryness, heat in an oven at 100° C. until of constant weight, and weigh.
10. Ash at red heat, cool, and weigh.

The coaly matter, or free carbon as it has been named in the tables, is given by the difference in weight between the residues from stages 9 and 10.

It is of interest to note that in the case of coal miners the analysis of the ash of the coaly matter separated from the lungs approximates very closely to that of the original coal in regard to the free and combined silica content. The results of analysis are shown in the accompanying tables.

Summary of Cases.

CASE I.—The patient was a coal miner, aged fifty-six, who died as the result of an accident. He had no known disability. He had been a coal miner in the northern collieries for thirty-five years.

Post mortem the lungs were slightly blackened; there was no nodular fibrosis or consolidation present. There was some peribronchial aggregation of coal dust in the upper lobes. The glands were greyish-black, soft and not enlarged.

CASE II.—The patient was a coal miner, aged fifty-six, who died as the result of an accident. He had no known disability. He worked in English collieries until 1911, and in Australian collieries from 1911 to 1931.

Post mortem there was a fracture of the spine; the heart and abdominal organs were normal. In the left

[1] Alcoholic potash: Potassium hydroxide, 20 grammes; water, 300 cubic centimetres; alcohol (95%), 600 cubic centimetres.

[2] Acid Alcohol: Hydrochloric acid, 100 cubic centimetres; water, 300 cubic centimetres; alcohol (95%), 600 cubic centimetres.

TABLE I.

Showing Milligrammes per Gramme of Dried Lung of the following Substances.

Class.	Case Number.	Free Carbon.	Ash.	Combined Silica.	Free Silica.	Alumina.	Ferric Oxide.	Calcium Oxide.	Magnesium Oxide.	Phosphoric Anhydride.	Remarks on Pathology of Lungs.
I	I	16·2	29·0	6·6	0·98	3·7	3·9	0·57	0·51	4·8	No nodules, no consolidations.
	IV	28·0	36·2	2·9	0·90	2·3	5·5	—	—	—	No nodules, no consolidations.
	VI	50·0	30·4	1·1	1·5	1·2	6·3	1·8	0·70	12·6	Few fibrotic nodules.
	XII	85·0	52·9	10·6	4·7	10·1	5·7	4·2	0·58	11·6	Consolidations and nodules.
	XIII	90·0	37·0	8·4	0·60	7·3	7·0	0·59	0·32	8·0	No nodules, no consolidations.
	XXII	111·8	88·5	10·2	0·32	9·4	3·6	26·8	1·6	28·6	Many nodules and consolidations.
	XXVII	96·0	23·8	0·90	1·3	1·4	5·5	1·4	0·47	10·6	Fibrotic nodules and consolidations.
	XXX	35·5	46·7	4·0	3·6	3·8	16·4	1·4	0·63	10·3	No nodules, no consolidations.
II	III	15·0	63·0	7·2	7·2	8·9	6·8	—	—	—	Small areas of consolidations.
	V	8·5	70·0	10·8	16·0	8·3	9·0	5·3	0·90	15·7	Gross consolidation cuirass type.
	VII	51·2	40·8	3·6	0·7	4·2	8·5	1·2	0·70	12·8	No fibrous nodules, no consolidation.
	X	13·2	55·5	14·3	5·8	8·8	8·4	0·89	0·72	11·4	Consolidation and fibrotic nodules.
	XXI	9·8	54·7	6·1	1·7	4·3	5·4	1·0	0·92	19·4	No fibrotic nodules, no consolidations.
	XXIX	81·5	32·4	1·5	2·1	1·8	4·2	3·3	0·61	11·7	Consolidations with cavitation and fibrotic nodules.
III	II	42·0	27·7*	1·9	0·52	4·7	3·5	—	—	—	
	VIII	74·5	37·5	2·4	3·2	3·6	4·4	1·2	0·71	13·6	No fibrotic nodules, no consolidations.
	XI	30·2	36·2	2·2	0·69	2·0	8·4	0·87	0·55	10·8	No fibrotic nodules, no consolidations.
	XXVIII	72·5	56·4	11·0	4·2	8·9	9·9	1·2	0·20	13·0	No nodules, no consolidations.
	XXXI	67·5	54·0	4·3	5·2	4·6	13·4	2·1	0·85	15·8	Consolidations and nodules.
IV	XVII	1·5	18·3	1·3	3·4	0·97	2·6	0·77	0·25	7·0	Many fibrotic nodules.
	XIX	3·0	21·1	1·9	7·0	1·5	4·9	—	—	—	Many fibrotic nodules.
	XXIV	5·8	31·8	3·1	5·6	1·6	7·3	2·1	0·42	10·8	Fibrotic nodules and consolidations.
V	XIV	8·5	81·8	12·1	8·2	9·0	6·2	15·2	0·95	20·5	Gross silicosis.
	XVIII	8·0	25·5	4·6	3·2	4·2	4·0	0·5	—	7·0	Small number of fibrotic nodules.
	XXIII	6·8	76·4	18·4	10·0	15·7	5·6	1·8	1·4	12·8	Massive consolidations.
VI	IX	5·5	27·8	1·1	4·6	1·3	4·9	1·2	0·54	10·0	Consolidations.
	XX	4·2	109·0	18·4	41·6	12·5	21·8	1·1	0·50	8·0	Consolidations.
VII	XV	4·0	23·5	1·1	0·82	1·4	2·8	0·56	0·31	12·2	No abnormality.
	XVI	3·0	30·2	0·87	0·27	0·45	4·4	1·5	0·80	10·3	Basal pneumonia.
VIII	XXV	2·0	20·2	0·22	0·34	0·48	1·6	3·6	0·49	10·2	Gross tuberculosis.
	XXVI	4·0	28·5	0·77	0·54	0·49	2·9	7·7	0·50	12·0	Gross tuberculosis.

lung generalized blackening was found. There were no areas of consolidation. No pleurisy was present. On section three small nodules were palpable. In the right lung generalized blackening was seen. The apex of the upper lobe had a few cicatrices and small consolidations over an area of five square centimetres. A few small black nodules were present in the lung. Both lungs were spongy with a little pneumonic consolidation at the bases.

On section the area of consolidation of the upper lobe consisted of fibroblastic tissue, dust cells and lymphoid tissue. One of the few small black nodules, about two millimetres in diameter, consisted of vestigial vascular or alveolar elements with dust cells and fibroblastic tissue and had not the appearance of a laminated silicotic nodule. Sections near the pleura showed small areas of fibroblastic tissue and lymphoid tissue, and large aggregates of dust cells. There were no laminated nodules; this condition is an example of the pleural drift of dust cells. The alveoli generally showed emphysema.

CASE III.—The patient had been a coal miner, a metalliferous miner and a steel worker. He was aged fifty-nine and died by drowning. He had no known disability. He worked in a colliery in the northern field for seven years; at Cobar he was copper mining for eight years; he was at Collie coalfields (Western Australia) for six years. He worked in the Western Australian goldfields, Murchison and Black Range, for eight years. He finished mining at the age of forty-three. He was employed in the steel works from 1914.

Post mortem, in the left lung generalized blackening was seen with sago grains on the surface. The upper lobe had a small consolidation of the pleural region which on section was six millimetres thick and extended over the surface of the lung with an adherent pleura. There were no definite nodules apart from sago grains. The right lung was generally blackened, sago grains were present in the lower and middle lobes. A small area of consolidation was seen in the apex of the upper lobe and another at the

TABLE II.

Showing Cummins and Sladden's Analyses and Notes of Lungs of Coal Miners Dying of Pulmonary Disease after Long Disablement, extracted from their Table and Text and Percentage Figures changed to Milligrammes per Gramme of Dried Lung.

Cummins and Sladden's Case Number.	Ash.	Silica, Free and Combined.	Coal Ash Free.	Remarks.
II	52·6	9·7	—	Lungs intensely fibrotic. Tuberculosis.
III	52·9	11·0	—	Intense fibrosis with nodules.
V	104·0	39·1	51	Intense anthracosis with nodules.
XIV	62·7	10·8	249	Intense fibrosis with nodules and cavities.
XV	103·4	12·7	77	Lungs fibrotic with nodules.
XXI	112·2	32·0	—	Areas of fibrosis with nodules.
XXVI	91·3	22·5	28	Anthracosis with cavities.
XXVIII	63·0	11·2	63	Anthracotic with nodules and cavity.
XXIX	89·6	37·9	13	Consolidation with nodules.
XXXII	124·2	42·8	110	Intense fibrosis with cavitation.
XXXIII	105·1	32·2	63	Fibrosis. Nodular tuberculosis.
XXXVIII	49·0	20·8	—	Consolidation with nodules.
Averages	84·2	23·6	82	

TABLE III.

Showing Cummins and Sladden's Analyses and Notes of Lungs of Coal Miners Not Dying of Pulmonary Disease and Not Known to have any Pulmonary Disability, extracted from their Table and Text and Percentage Figures changed to Milligrammes per Gramme of Dried Lung.

Cummins and Sladden's Case Number.	Ash.	Silica, Free and Combined.	Coal Ash Free.	Remarks.
I	44·0	7·6	—	Advanced anthracosis with nodules.
IV	57·6	10·3	—	No special signs of anthracosis.
VII	64·0	8·7	55	Mild degree of anthracosis.
VIII	52·1	5·5	80	No high degree of fibrosis or anthracosis.
IX	79·0	15·0	131	Moderate anthracosis.
X	71·0	12·0	67	Lungs black and œdematous.
XII	66·4	14·5	206	Nodules in upper lobes.
XIII	61·0	13·2	52	Nodules at apices.
XVII	61·4	10·4	49	Right apical lobe anthracotic and nodular.
XX	115·0	40·7	15	Apical lobes fibrotic, anthracotic and nodular.
XXII	80·0	17·8	172	Anthracosis moderate.
XXIII	83·0	19·1	216	Anthracosis apical lobes.
XXIV	65·0	6·4	36	Fibrosis and cavitation of right apex.
XXV	48·0	7·7	37	No disease to naked eye.
XXX	48·0	4·2	22	Apical lobe anthracotic with cavitation.
XXXI	47·0	5·2	54	Mild anthracosis.
Averages	65·2	12·4	85	

base of the lung. On section no definite nodules, apart from sago grains, were seen. Consolidations were about two centimetres square and extended over the surface of the lung.

On section of the lungs sago grains of the surface showed a structure typical of sago grains in silicotic lungs. The consolidations showed typical laminated nodules of silicosis, but were surrounded by areas of fibrous tissue, cellular and non-cellular, containing numerous dust cells and occasionally masses of lymphoid tissue. The alveoli showed emphysematous changes and a good deal of thickening by fibroblastic tissue, with numerous dust cells.

CASE IV.—The patient was a coal miner, aged seventy-seven. He died at work. Nothing is known of his industrial history before 1866. He worked in collieries in the northern field for twenty-nine years.

Post mortem pleurisy with effusion, chronic nephritis and a dilated heart were found. The lungs were blackened. Large bullæ and well marked adhesions were present. There was a marked condition of dilatation of the alveoli. Peribronchial deposition of coal dust was not very marked.

CASE V.—The patient had been a metalliferous miner and a coal miner; he was fifty years of age. A year before death he had complete disability from dyspnœa and debility. The sputum was examined without success for tubercle bacilli. X ray examination revealed massive fibrosis of both lungs.

He was a surface worker in the Occidental gold mine (Cobar) for five years and a miner for seven years; he worked at the Gladstone copper mine (quartz), Cobar, as a miner for two years; at Ardlethan tin mines for one year; in a western field colliery, shaft sinking and general coal mining, for nine years. From this history it appears that this man was exposed to mine dust of silicates with a varying percentage of free silica for about nine years in Cobar and the Ardlethan mines. There followed then a period of shaft sinking in a western colliery in which the shaft was sunk through sandstone for twenty-five feet. For three months he worked with dry drills in a sandstone drive, and for one year doing similar work with wet drills. There is thus a history of less than two years' sandstone work, of nine years' Cobar mining, followed by eight years' general coal mining work.

Post mortem the right lung was covered on the pleural surfaces with very dense inflammatory tissue. Outer portions of the lung were densely consolidated—the cuirass type of consolidation. On section the whole of the upper lobe, except the medial lower portion, was of a dense black colour and rubbery consistence, and the consolidation showed no pattern. This consolidation extended as a sheath one inch thick over the middle and lower lobes on the outer aspects. The unconsolidated portion of the lung was blackened with peribronchial deposits of fibrous tissue and dust cells, but there were no fibrotic nodules. The glands were dense, black and rubber-like. The left lung showed similar characters. The consolidated areas consisted of dense fibrous masses, containing numerous collections of dust cells. Neither tuberculous tissue nor tubercle bacilli were found in sections (see Figure I).

CASE VI.—The patient was a coal miner. His age was unknown. He died while at work and had no known disability. He worked in a northern field colliery for twenty years.

Post mortem examination revealed general atheroma of the arteries and a dilatation of the right side of the heart, which was considered to be the cause of death. The lungs were blackened. A giant bulla the size of a small fist was present in the upper lobe of the right lung, with several smaller but still very large bullæ. A number of pin-head white areas were present on the surface of the upper lobe. The upper lobe had puckered areas and contained a number of small irregular black nodules three to five millimetres in diameter. The alveoli were dilated. There was a definite pleural drift of dust cells with subpleural thickening. The lower lobe showed few changes. The glands were soft and black. The left lung showed similar changes.

CASE VII.—The patient was a coal miner and had been a metalliferous miner. He was seventy-four years of age. He died of infective cholangitis. He was born in Scotland and worked in mines near Edinburgh. He came to Australia in 1884 and worked in northern field collieries until three years before his death. He worked for three years in gold mines in Western Australia. In all he worked in mines for sixty years.

Post mortem examination revealed degenerative changes in arteries and infective cholangitis. The lungs were blackened; there was pneumonic consolidation of the lower lobes. There were no fibrous nodules or fibrous consolidation. There was a good deal of peribronchial deposition of coal dust, with some dilatation of the alveoli; there was no marked pleural drift of dust cells.

CASE VIII.—The patient was a coal miner, aged fifty-four. He died suddenly; there was no history of disability. It was suggested that the cause of death was angina. He wore a mustard plaster in the precordial region. He came to Australia from England six years ago and worked in a northern field colliery.

Post mortem the right lung was generally blackened; there were no adhesions. A few doubtful sago grains were present in the pleura of the upper lobe. There were no nodules or consolidation. Peribronchial coal dust deposition was not marked. The glands were soft and black. Left lung appearances were similar.

CASE IX.—The patient was a foundry dresser and annealing pot cleaner, aged fifty-two. There was a history of debility, loss of weight and cough for four years. A radiograph revealed pulmonary fibrosis. No tubercle bacilli were found in sputum in several examinations. He was a groom in a London bus company until 1909, a station hand in New South Wales until 1920, a foundry dresser in a steel foundry for seven years. His duties were to knock sand off castings with a percussive machine and also to work inside annealing pots knocking sand off with a percussive machine and a grinder. An annealing pot is about four feet six inches in diameter by six feet six inches in length. This work was extremely dusty. No protective breathing apparatus was worn.

Post mortem the left lung had a pleura much thickened and adherent generally. There was a large cavity in the upper lobe; the cavity was smooth, black walled and crossed by trabeculæ. It took up nearly the whole of the upper lobe and its ramifications extended in areas of consolidation only. In the lower lobe the consolidation was of the cuirass type, one inch thick. The medial portion of the lower lobe was soft; there were no fibrotic nodules.

In the right lung the pleura was dense and adherent generally. The upper lobe was mostly a massive irregular consolidation. The consolidated areas had small cavities with or without gelatinous contents. The lower lobe presented in part a similar character.

Histologically the lungs showed massive fibrosis of a dust type; there was nothing suggesting tuberculous infection; the walls of the cavities, large and small, showed necrotic changes.

FIGURE I (Case V).

Left lung of a metalliferous and coal miner, showing almost complete consolidation of upper lobe. The consolidation shows no nodulation and has a cuirass-like extension over the exterior of the lower lobe, an example of silico-anthracosis.

CASE X.—The patient had been a coal miner and a metalliferous miner. He was forty-five years of age. He had had recurrent attacks of bronchitis with pleurisy for the past ten years of life. Repeated sputum examinations failed to reveal tubercle bacilli. The radiograph showed well marked pneumonokoniosis. He died from cardiac failure two and a half years after leaving work with severe dyspnœa. He did timber cutting and bush work for some years. He was a surface worker at Mount Lyell for two years and at Bendigo gold mining for four and a half years. He was in the northern field coal mining for a period of twenty-one years.

Post mortem a partial right pneumothorax was present; the lungs were very bullous along the anterior margins, markedly adherent, with pleurisy and congested bases. The right lung was covered with adhesions and a few sago grains could be seen. The exterior of the upper lobe presented a ragged appearance, probably due to tearing of adhesions. The upper lobe was the site of an irregular consolidation involving nearly the whole lung. The mutilation might in part have been due to anthracotic cavitation. There were no areas of caseation; the lower lobe changes were much less marked. There were a few one to two millimetre black nodules in relation to peribronchial tissues. The left lung had well developed adhesions, but a few sago grains could be made out at the base. There was a very large bulla at the margin of the upper lobe; there was an area of irregular consolidation in the lower part of the upper lobe; the apex of the lobe was puckered, fibrous and cystic; the alveoli were dilated. There were no nodules or consolidations in the lower lobe and the peribronchial deposition of coal dust was not so marked. There was no evidence of tuberculous changes.

CASE XI.—The patient was a coal miner, aged forty-eight. He was electrocuted; he had no known disability. He worked in English collieries from 1896 to 1907. He worked in northern, western and south coast fields for a period of twenty-three years.

Post mortem the right lung was greyish-black, with a few pleuritic adhesions of the upper lobe. There were no sago grains, no consolidations, and no nodular fibrosis. The peribronchial dust deposition was not marked. The left lung presented similar appearances.

CASE XII.—The patient was a coal miner, aged fifty-two. Two years before death he complained of shortness of breath for some years, pleurisy of a few weeks' duration, night sweats, cough with sputum and loss of weight. Clinically, bronchial breathing was present in both upper lobes, with crepitations. The radiograph shows massive consolidation of the right upper lobe and smaller consolidations in the left upper and lower lobes. There was a pneumothorax at the apex of the left upper lobe, with generalized fine fibrosis and a centrally placed heart. He worked in a slate quarry in England for eleven years, in south coast pits for twenty-seven years. He worked for three years in headings through sandstone and shale.

Post mortem the right lung was covered with adhesions, which were very massive over the upper lobe. The lung was generally blackened. The whole of the upper lobe was one mass of consolidation and was the site of a very large area of cavitation and of mutilation due to tearing of adhesions. The cavitation appeared in part anthracotic and in part tuberculous. Where the consolidation had not been eaten away, it was dense, black and rubber-like. The glands showed areas of commencing caseation. They were large; some were hard and some were soft. The bases of the lower lobe showed areas of whitish consolidation, probably tuberculous bronchopneumonic patches. The lower lobe showed about a dozen nodules from two to ten millimetres in diameter, chiefly subpleural.

The left lung showed marked adhesions, particularly over the upper lobe, which had a large consolidation involving its greater part. This was the site of an anthracotic cavitation. At its apex was a small area of soft tissue with numerous pin-head areas of caseation. The cavitation ramified through the area of consolidation. There were a few fibrotic nodules in the lower lobe. The coal dust accumulation in the lower lobe was not nearly so marked as that of the upper lobe. The area of soft tissue with pin-head areas of caseation showed on section tuberculous tissue and tubercle bacilli.

CASE XIII.—The patient was a coal miner, aged fifty-four. He died at work. The radiograph shows a very minor degree of fine fibrosis. He worked in a London underground for four years tunnelling through clay, in a south coast colliery (New South Wales) for sixteen years coal mining, and for five years as a yard hand. He was two years off work suffering from nystagmus.

Post mortem there was atheroma of coronary arteries sufficient to account for sudden death. The right lung was

generally blackened, with adhesions of the base. There were no fibrous nodules or consolidations. Peribronchial deposition of dust cells was not unduly marked. There was no evidence of pleural drift. The left lung presented similar characteristics.

CASE XIV.—The patient was a metalliferous miner, aged fifty-two. The cause of death was silicosis and tuberculosis. The radiograph shows generalized coarse nodular fibrosis with massive consolidation in the right, middle and upper lobes. Smaller consolidations and a large cavity were present in the left lower lobe, with marked adhesions of the diaphragm. He worked as a quartz miner in Victoria for two years; at Broken Hill (machine drilling) for one year; at Kalgoorlie (machine drilling) for fourteen years; as a sewer miner, Sydney, for two years; as a sewer miner, Canberra, for five years.

Post mortem the left lung was covered with gross adhesions. The whole lung was bulky and very hard. It was composed almost entirely of small and large nodules which had here and there fused into areas of consolidation. There was a very large tuberculous cavity in the upper portion of the lower lobe, together with many areas of caseation, particularly in the lower lobe (see Figure II). The glands were very large, greyish-black and very hard.

The right lung presented a similar appearance. There was no tuberculous cavity in this lung, but the middle lobe was consolidated, with many areas of caseation. The appearances of the lungs were those of gross silicosis and tuberculosis.

CASE XV.—The patient was a railway worker and storekeeper, aged fifty. There was no known exposure to dust. He died from cyanide poisoning.

Post mortem the lungs showed no abnormality.

CASE XVI.—The patient was a dealer, aged forty-three. He had no known exposure to dust. He died from a fractured skull and terminal pneumonia.

Post mortem the lungs showed basal pneumonia.

CASE XVII.—The patient was a stonemason, aged fifty-one. The radiograph shows a coarse nodular fibrosis with consolidations of both upper lobes. Tubercle bacilli were found in the sputum. He worked as a stonemason in Sydney sandstone for twenty-six years.

Post mortem the lungs were covered with dense adhesions; the upper lobes of both lungs had large tuberculous cavities. There were numerous fibrotic nodules from one to five millimetres in diameter, more frequent in the upper lobes. The glands were soft, greyish, with small areas of caseation. There were small areas of caseation in the lower lobes.

CASE XVIII.—The patient was a metalliferous miner, aged forty-five. He died suddenly from coronary disease of the heart. He was quartz mining for an indefinite, but probably short, period some years before he died.

Post mortem the lungs were greyish black. There were a number of sago grains, more frequent in the lower lobes. In the upper lobes there was a small number of fibrous nodules of one to three millimetres in diameter. The lower lobes contained no nodules. The glands were small, not very hard, and greyish.

CASE XIX.—The patient was a stonemason, aged forty-nine. The cause of death was tuberculosis. The radiograph shows a coarse nodular fibrosis with consolidation at both bases. He was a Sydney sandstone mason for many years.

Post mortem the left lung was greyish, with few adhesions; sago grains were scarce. There were a number of fibrotic nodules, one to three millimetres in diameter, in the upper lobe. At the base of the upper lobe there was an area of caseation and tuberculous bronchopneumonia. There were a few nodules in the upper part of the lower lobe. The glands were greyish, enlarged and hard.

In the right lung there were a few adhesions; sago grains were not obvious. The upper lobe had a number of fibrotic nodules one to three millimetres in diameter. These were also present in the upper part of the lower and middle lobes. The glands were enlarged and very hard. There was some perigiandular caseation.

Sections show laminated whorled nodules free from infective changes, and necrosed composite nodules surrounded by fibroblastic tissue, characteristic of tuberculous changes in silicotic nodules.

CASE XX.—The patient was an ore miller, aged fifty-two. The cause of death was pneumonokoniosis, pneumothorax and cardiac failure. There had been complaint of cough, pain in the chest, and dyspnœa. The radiograph shows an advanced degree of pneumonokoniosis and a very marked pneumothorax of the right side. He worked at ore milling for eight years and was a farmer before this. He crushed quartz, hæmatite, limestone, talc, ochres, kaolin, fullers' earth and cornish stone, and was gravely exposed to dust at this occupation.

FIGURE II (Case XIV).

Left lung of a metalliferous miner, showing large and small fibrotic nodules and consolidations with areas of caseation and a large tuberculous cavity in a lung illustrative of gross silicosis and tuberculosis.

Post mortem, when the right pleural cavity was opened, air escaped under some pressure. Right pneumothorax was present and the right lung collapsed to some degree. Numerous dense adhesions were present at both apices, irregularly scattered over lateral and posterior surfaces of both lungs and in interlobar regions. Both lungs showed large patchy areas, yellowish in colour and quite firm, occupying a considerable portion of lung tissue. Marked emphysema (interstitial) was present in both lungs, some areas showing thin-walled bullæ standing well out from the surface. Sections of the solid areas sank in water. Numerous yellowish enlarged mediastinal glands were present. There was no evidence of tuberculosis. Slight tracheitis and bronchitis were found. The left and right ventricles were dilated and hypertrophied. Some atheroma of aorta and coronary arteries was present.

Examination of the lungs after preservation showed that they were reddish, all parts being heavily pigmented with oxide of iron. Throughout the lungs there were large areas of reddish consolidation somewhat irregular and not having any nodular structure. This development of consolidated areas was most marked in the upper lobes. The apex of the right upper lobe had a subpleural area of greyish-black consolidation in which a nodular character could be discerned. The glands were reddish, not greatly enlarged and not very hard. There were no definite tuberculous changes.

CASE XXI.—The patient was a coal miner and a metalliferous miner, aged forty-nine. He died from a cerebral tumour. He worked at metalliferous mining at Broken Hill and Cobar for six years, and as a coal miner on the western field for nine years.

Post mortem the right lung was greyish-black. There were no fibrotic nodules and no consolidations. The glands were black, small and soft. The left lung was covered with adhesions. There were no consolidations and no fibrotic nodules. The glands were black and soft. There was very little coal dust in this lung.

CASE XXII.—The patient was a coal miner, aged fifty-two. The cause of death was a failing heart. The radiograph shows generalized coarse nodular fibrosis with consolidations in the upper and lower lobes of both sides (see Figure III). He worked for thirty-four years in the same South Coast coal mine, twenty-eight years as a miner at the face, two years as a wheeler, and four years on the surface at the screens.

Post mortem the lungs were generally blackened, with small adhesions and well marked bullæ in the upper lobes. In the right lung very hard subpleural nodules and pleural surface. There centimetres in diameter in the upper portion of the upper lobe. It was composed of separate nodules. Distributed throughout the lung were nodules up to five millimetres in diameter. In the lower lobe these were chiefly subpleural. There was a nodular consolidation at the base of the right upper lobe. The lung was very emphysematous. The lung tissues were charged with coal dust (see Figure IV).

FIGURE IV (Case XXII).

Right lung of a coal miner, South Coast colliery, showing consolidation made up of fibrotic nodules in the apex and base of upper lobe with generalized fibrotic nodules and emphysematous changes. The condition is nodular pulmonary fibrosis not due to free silica.

In the left lung there was a large area of consolidation at the upper part of the upper lobe. It was made up of a large number of loosely aggregated nodules. Excepting the lower lobe, the development of fibrous nodules in the lung was very marked. There was also a large consolidation made up of a fusion of a number of separate nodules at the base of the right upper lobe. The subpleural position of many of the nodules was similar to that described for the right lung. The lung was heavily charged with coal dust (see Figure V).

FIGURE V (Case XXII).

Left lung of a coal miner, South Coast colliery, showing similar characters to right lung (see Figure IV).

There was no evidence of tuberculous infection. The glands were black, soft and not enlarged.

CASE XXIII.—The patient was quartz mining at Hill End and Padylackey and mining in serpentine at Lucknow for twelve years. He worked at a cement works for nineteen years.

Post mortem the left lung was solid. It was covered with adhesions from four to five millimetres thick. The upper lobe was a massive consolidation in which a nodular character could be made out. The lower lobe was a mass of fibrotic nodules two to five millimetres in diameter. Areas of caseation occurred through the lung and there were numerous large tuberculous cavities. The glands were large, soft, and showed early caseation (see Figure VI).

The right lung was covered with adhesions. The upper lobe was the site of a massive consolidation which showed a nodular character and had areas of caseation. The rest of the upper lobe was made up of large fibrotic nodules and large areas of caseation. The lower and middle lobes have numerous nodules, two to five millimetres in diameter, and areas of caseation (see Figure VII).

CASE XXIV.—The patient was a stonemason, aged sixty-eight. The radiograph shows coarse nodular fibrosis of the right lung with consolidation in the hilar region and the lower lobe. The left lung shows coarse nodular fibrosis with consolidations in the upper and lower lobes. There was a history of cough with sputum. Tubercle bacilli were not found in the sputum. He worked as a stonemason in Sydney sandstone for seventeen and a half years. Elsewhere he worked on mixed stone for nine years.

Post mortem the right lung only was obtained. The lung was grey. There were some adhesions. Sago grains were not obvious. The upper middle and to a lesser extent the lower lobe had a number of fibrotic nodules ranging in size from two to five millimetres. There was an aggregation of nodules forming a well marked consolidation in the middle lobe. There were several smaller consolidations of nodules in the upper lobe. There was no obvious tuberculosis. The glands were hard and slightly enlarged.

CASE XXV.—The patient was an ironworker, aged forty-six. The cause of death was chronic phthisis. There was a three years' history of ill health, cough and loss of weight. The sputum contained tubercle bacilli. The radiograph shows, in both lungs, gross fibrous tuberculosis with cavitation. He was an ironworker in a foundry for many years.

Post mortem both lobes of the left lung showed generalized massive adhesions, with gross cavitation and caseation. The right lung showed generalized massive adhesions. The upper lobe showed gross cavitation and caseation. The lower lobe showed areas of caseation and tuberculous bronchopneumonia.

CASE XXVI.—The patient was a salesman, aged fifty. The cause of death was chronic phthisis and auricular fibrillation. There was a ten years' history of chronic phthisis. He was for seven years a sanatorium inmate.

Post mortem the left lung was covered with adhesions.

The upper lobe was the seat of chronic phthisical changes and very little of it remained. The lower lobe had a few areas of caseation in its upper part.

In the right lung there were generalized adhesions, especially of the upper lobe. The upper lobe was the seat of chronic phthisical changes and very little of it remained. The middle lobe showed caseation and fibrosis, the lower lobe small areas of caseation.

CASE XXVII.—The patient was a coal miner, aged sixty-two. The cause of death was pneumonia. The radiograph shows coarse fibrosis with a large area of consolidation in the left upper lobe and a small area in the right. He was engaged in bush work and coal mining in the Lithgow district all his life. He worked for the last twenty-one years in a western field colliery.

Post mortem the left lung was densely black. There were greyish-white areas on the surface of both upper lobes, the site of flattened subpleural consolidations. The upper lobe had at its apex an area of consolidation four centimetres in diameter, largely excavated by non-tuberculous anthracotic cavitation. There were a number of fibrotic nodules one to two millimetres in diameter. The upper portion of the lower lobe had many nodules two to five millimetres in diameter, with dilated alveoli, recalling the appearances seen in Case XXII. Both lobes were densely black. The glands were black and soft and not enlarged (see Figure VIII).

In the right lung upper and middle lobes were massive and œdematous, with pneumonic consolidation. The apex showed small consolidations with subpleural plaques with cavitation. Nodules of two to five millimetres were present throughout the upper part of the lung. The lower lobe was densely pigmented. There were a number of small fibrotic nodules one to two millimetres in diameter, mostly subpleural. The glands were black, soft and not enlarged.

FIGURE VI (Case XXIII).
Left lung of a quartz miner and cement worker. The upper lobe shows a massive consolidation in which a nodular basis can be made out. The lower lobe shows fibrotic nodules and areas of tuberculous caseation.

FIGURE VII (Case XXIII).
Right lung of a quartz miner and cement worker, showing fibrotic nodules in upper and middles lobes with areas of tuberculous caseation.

FIGURE VIII (Case XXVII).
Left lung, upper lobe, of a coal miner, western field, showing anthracotic consolidation with non-tuberculous cavitation, with many fibrotic nodules and well marked emphysematous changes.

CASE XXVIII.—The patient was a coal miner, aged sixty-five. He died suddenly at work; he had been treated for *angina pectoris*. He came from Scotland nine years ago and worked in northern collieries.

Post mortem nodular atheroma of the coronaries with thrombosis was found. The right lung was generally blackened. There were no fibrotic nodules present and no consolidation. The upper lobe was charged with coal dust; the lower lobe contained much less. The glands were soft and black. The left lung presented a similar appearance.

CASE XXIX.—The patient was a coal miner, aged seventy-one. The cause of death was cardiac disease. He died at work. Auricular fibrillation was diagnosed ten days before death, and he was advised not to return to work. He had pneumonia and bronchitis one year before death. He did surface work at Cobar copper mines for three years, and he was prospecting in various goldfields for seven years. He was shaft sinking at Lucknow (serpentine) for one year. He worked for twenty-six years at a western field colliery.

Post mortem the right lung was very black. There was a large consolidation like black india-rubber in the upper lobe, with an early degree of anthracotic cavitation. There was a large number of fibrotic nodules two to three millimetres in diameter in the upper lobe, which was intensely black. Subpleural plaques of consolidation were present.

The left lung was intensely black. There were subpleural plaques and a large consolidation in the upper lobe which showed early non-tuberculous anthracotic cavitation. There were fibrotic nodules at the periphery of the consolidation and the rest of the upper lobe

contained a large number of fibrotic nodules two to three millimetres in diameter. The lower lobe had a small area of consolidation at its apex and a small number of fibrotic nodules in its upper part. The upper lobe was heavily charged with coal dust. This was not so marked in the lower lobe (see Figures IX and X). The appearances of these lungs are similar to those seen in Cases XXII and XXVII.

Case XXX.—The patient was a coal miner. He died following an accident. His age is unknown. The industrial history is not available.

Post mortem the right and left lungs were greyish and œdematous. There were no fibrotic nodules and no consolidations. Peribronchial dust depositions were seen in the upper lobes, with emphysematous changes. The glands were not enlarged. They were soft and black.

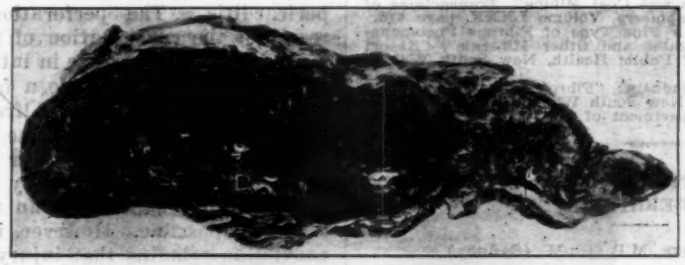

Figure IX (Case XXIX).
Left lung, upper lobe, of a coal miner, western field, showing well marked anthracotic consolidation with non-tuberculous cavitation.

Case XXXI.—The patient was a coal miner, aged fifty-seven. The cause of death was coronary occlusion. The radiograph showed fine fibrosis with small apical consolidations. There is a history of attacks like asthma. He was born in Scotland and began work at the age of nine years. He worked in hard headings until fifteen years of age. He worked in New South Wales northern collieries for twenty-eight years.

Post mortem the aorta and coronary arteries showed a condition of gross atheroma sufficient to account for his sudden death. In the right lung consolidation was found at the apex, with a diameter of three centimetres, which joined with a subpleural consolidation two millimetres thick; this extended for several centimetres. The upper lobe showed the peribronchial tissues charged with coal dust, and, except at the very apex, the soft lung tissue was greyish. The middle lobe had a few flattened subpleural nodules; the lower lobe showed few changes.

In the left lung the upper part of the upper lobe showed a subpleural thickening or consolidation up to five millimetres in thickness, which extended over an area of a few centimetres and showed several minute anthracotic cavitations. There were also a few other subpleural nodules and about half the upper lobe was covered with subpleural consolidation one millimetre thick. The lower lobe showed a marked subpleural deposition of coal dust with development in the upper part of the lower lobe of subpleural consolidation extending over some centimetres and being one millimetre in thickness, together with a deeper irregular area of consolidation about thirty by five millimetres.

In both lungs the bronchi were filled with mucus.

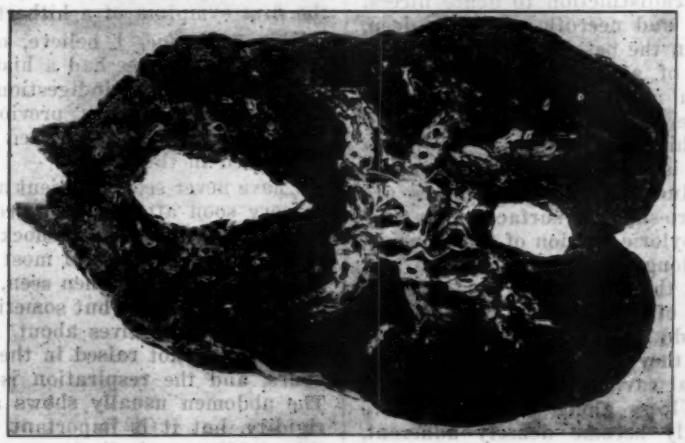

Figure X (Case XXIX).
Left lung, upper lobe, of a coal miner, western field, showing section of lung deeper than Figure IX. The section is opened out, the consolidation with cavitation is shown, and many fibrotic nodules and emphysematous lung changes.

Classification.

The following classification may be adopted for the cases described:

Class I.—Coal miners, New South Wales, with no metalliferous mining history and no known exposure to free silica: Cases I, IV, VI, XII, XIII, XXII, XXVII, XXX.

Class II.—Coal miners with a metalliferous mining history and known exposure to free silica: Cases III, V, VII, X, XXI, XXIX.

Class III.—Coal miners who worked also in pits outside Australia: Cases II, VIII, XI, XXVIII, XXXI.

Class IV.—Sydney sandstone masons: Cases XVII, XIX, XXIV.

Class V.—Metalliferous miners exposed to free silica: Cases XIV, XVIII, XXIII.

Class VI.—Workers in dusty occupations: Cases IX, XX.

Class VII.—Industrial workers with no known dust exposure: Cases XV, XVI.

Class VIII.—Individuals not exposed to dust, dead of tuberculosis: Cases XXV, XXVI.

Summary.

The industrial history, chemical analysis and pathology of the lungs of thirty-one persons are given, of whom nineteen worked as coal miners, some of whom had coal miner's lung and ten had pulmonary fibrosis due to their various occupations.

A method of analysis was employed by which it is considered that the free silica, not previously determined in any series of lungs, was correctly estimated.

A case of nodular pulmonary fibrosis in a coal miner not due to free silica, but to the carbon or the combined silica of coal dust (silicates), is described.

A case of non-tuberculous cavitation in silicotic fibrosis in a foundry worker is described.

The essential features of coal miner's lung—upper lobe consolidation, fibrotic nodules, non-tuberculous cavitation and coal dust accumulation as found in various lungs—are described.

A criticism of radiological practice is made, and a discussion on medico-legal aspects is given.

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PEFORATED PEPTIC ULCER: A REVIEW OF THIRTY-EIGHT CASES.[1]

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Perforation of a peptic ulcer is a most dangerous complication, and within a very short time the patient, from being in ill health, is in a condition of deadly peril; at the same time it is a common condition and one which demands immediate surgical intervention with a minimum of transportation, and therefore any medical man must be prepared to undertake its treatment.

Ulcers which perforate are always of the chronic type, that is, in contradistinction to acute ulcers, which are superficial and necrotic, they are deep, the muscular layers in the base are destroyed, and the floor is formed of adult fibrous tissue; the margins also contain much fibrous tissue, which gives the ulcer the characteristic raised and indurated edge. This fibrous tissue indicates attempted repair and is a measure of chronicity.

The commonest situations for these ulcers are the anterior and antero-superior surface of the duodenum, pylorus and pyloric portion of the stomach, and about midway along the lesser curvature. In the majority of cases the ulcer which perforates is found within an inch of the pylorus. Because they lie anteriorly they do not form adhesions, and when they rupture they contaminate widely an unprotected peritoneal cavity and give rise to the classical picture. Ulcers along the lesser curve much more frequently become densely adherent, usually to the pancreas; and the ulcerative process may penetrate the whole thickness of the stomach wall and involve that viscus without any leakage. When such an adherent ulcer perforates, it is usually at the extreme anterior edge, producing the usual symptoms.

Perforation into the lesser sac is very rare. The actual opening is usually small, about the size of a match head, or smaller, and the amount of gastric contents which escape is not large and the peritoneal cavity is not flooded, as is usually stated; most of the fluid present in early cases is peritoneal exudate and is protective in nature. The widespread board-like rigidity does not mean equally widespread peritoneal contamination, but is an intense reaction to an intense stimulus. It is found not only when other hollow viscera rupture, such as bladder or bowel, but in acute cholecystitis, acute appendicitis and ruptured hydatid cyst. I have seen extensive, but not universal, rigidity in acute pericarditis. The perforation is primarily the result of the continuation of ulceration, but there is no doubt that increase in intragastric tension due to food and drink is also a factor at times. One case at the Royal Prince Alfred Hospital occurred in a very drunken man whose stomach was washed out. The effect of muscular exertion and injury are debatable. One authority has attempted lately to prove that rupture of an ulcer from injury is almost impossible. However, in spite of proof, the conviction remains that injury cannot be regarded as a negligible cause. One case in this series, to be considered later, is very suggestive.

The commonest mode of onset is that the patient who has suffered for a long or short time with the chronic ulcer type of indigestion is suddenly seized with agonizing pain in the epigastrium. It is nearly always so definite that the time of rupture can be accurately fixed. Some patients will deny that they have had any previous indigestion at all, but when they are convalescing further questioning will usually elicit a definite history. However, there is no doubt that occasionally perforation is the first symptom of a hitherto silent ulcer.

This was true, I believe, of two cases of this series; twenty-three had a history of three months or more of previous indigestion, seven had a history of under three months' previous indigestion, three were too ill to be questioned sufficiently. It was not stated in three.

I have never seen a patient at the time of rupture or very soon afterwards. Probably they all show at first some degree of shock. By the time they are admitted to hospital most have recovered from any initial shock. When seen, they mostly lie still, moaning with pain, but sometimes are very restless and throw themselves about. The pulse and temperature are not raised in the first eight or twelve hours, and the respiration is thoracic and jerky. The abdomen usually shows a uniform board-like rigidity, but it is important to realize that often this rigidity markedly lessens after some hours and may, by comparison with the other side, be found in one rectus muscle only. This does not mean that the rupture is sealed over and that leakage has stopped. It has been well termed "the stage of dangerous improvement" and is only temporary, and if operation is delayed, will progress to the stage of toxæmia and peritonitis. Sometimes the onset is just as acute, but after a certain amount of leakage has occurred, the opening is occluded, the acute symptoms subside and localization occurs. In this way may be formed subphrenic, perigastric or pelvic abscess.

[1] Read at a meeting of the Western District Medical Association at Parkes, New South Wales, on December 14, 1932.

ILLUSTRATION TO THE ARTICLE BY DR. CHARLES BADHAM AND MR. HAROLD BURFIELD TAYLOR.

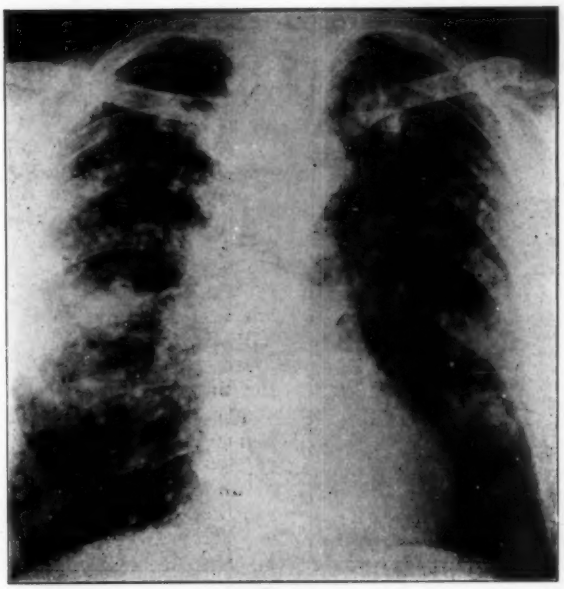

FIGURE III.
Radiograph of Case XII.

ILLUSTRATIONS TO THE ARTICLE BY DR. R. M. GLYNN.

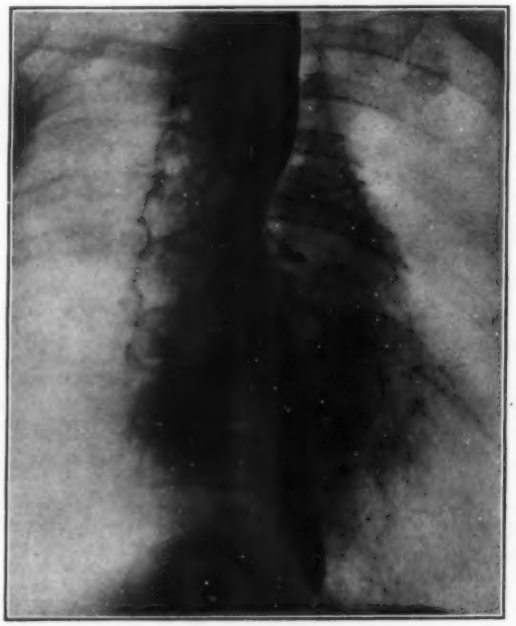

FIGURE I.
Showing barium filling traction diverticulum of the œsophagus.

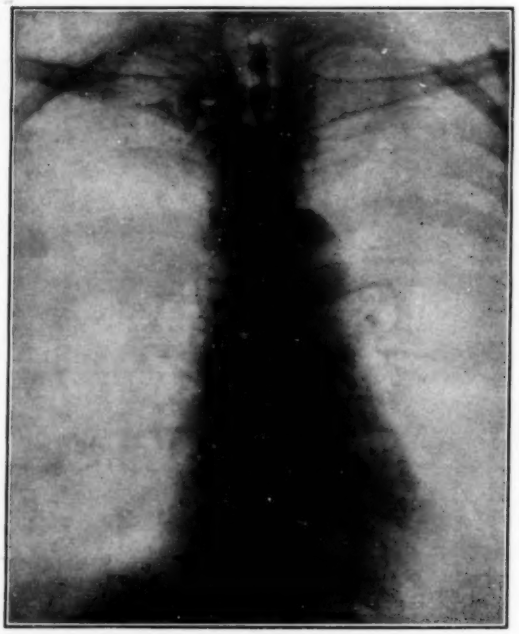

FIGURE II.
Showing barium in the left bronchus.

One patient in this series illustrates this form of onset.

His first admission to hospital was for a large perigastric abscess, which had formed after a typical acute onset. His second admission was for a ruptured gastric ulcer, close to the pylorus, eighteen months later.

Another case lies between the two modes of onset.

A man of fifty-eight had had sudden severe abdominal pain five days before, which had gradually settled down. Before admission to hospital he had two more similar attacks. He was very ill with general peritonitis, and had an ulcer at the back of the pylorus, just at the epiploic foramen, which was no longer leaking. After a rather stormy convalescence he recovered.

The progress of the disease is, with few exceptions, towards general peritonitis and toxæmia, with which I do not wish to deal. Probably now and then a patient with a small perforation and small leakage will recover without operation and with the condition undiagnosed.

Of the 38 ulcers in the series, 17 were gastric and 21 duodenal. Usually the proportion of duodenal ulcers is much higher. There were 35 males and 3 females, as shown in Table I.

TABLE I.

Showing Localisation and Sex Incidence.[1]

Sex.	Gastric Ulcer.	Duodenal Ulcer.
Males	14	21
Females	3	0

[1] The average age was 43·6 years. The youngest was 22 and the oldest 87.

Treatment.

It is essential that the opening should be closed in all cases. I have seen two patients treated by drainage only; both developed duodenal fistulæ and died. The incision is a matter of choice. A rapid one is through the *linea alba*, which can be closed with through-and-through silkworm gut sutures; after closing the peritoneum the sutures are tied over an anchored dressing.

The perforation can be closed in whatever way the surgeon wishes. The important thing is to get peritoneal apposition, which usually means a second row of Lembert sutures. Reinforcement of the suture line with an omental tag is advisable, but probably not essential. Is anything more than simple suture necessary or advisable? This has been debated for many years, and there is still no uniformity of opinion. If we admit that the ideal method of attack on chronic ulcer has not yet been settled, and if we consider that the rupture needs urgent treatment while the ulcer itself does not, then simple suture alone will be the method of choice. But there are occasional ruptured ulcers which from their size and position cannot easily be closed by suture. In early cases more radical treatment can be carried out safely, and I believe these are the only cases in which anything more than simple suture should be done.

One patient, a female, forty-two years of age, had such an ulcer high on the lesser curvature. The induration was about 5·0 centimetres (two inches) in diameter and the perforation could not be closed; the ulcer was excised. Five years later she was found to have an hour-glass stomach with an ulcer penetrating the pancreas, which was successfully treated by partial gastrectomy.

Probably a primary partial gastrectomy would have been the correct treatment.

A man of forty-two was operated on for a ruptured gastric ulcer close to the pylorus which was closed by suture. Three months later his symptoms returned; he had a fractional test meal and was on his way to hospital for a barium meal when his ulcer ruptured a second time. Operation revealed an ulcer surrounding three-quarters of the circumference of the pylorus. It obviously could not be sutured again, and a partial gastrectomy was carried out safely.

Excision of the ulcer, suture with primary gastro-enterostomy and partial gastrectomy, have all been strongly recommended by various surgeons in Europe and America for the treatment of ruptured ulcer. In the hands of experts dealing with large numbers of cases these procedures have not carried any added mortality, but I feel sure that most of us would avoid anything more than simple suture except when special indications existed.

The immediate mortality depends primarily on the number of hours which elapse between rupture and operation; and secondarily on the age of the patient. The old people stand this catastrophe very badly. The published statistics have shown rates which vary tremendously. Probably the average death rate is over 20%. The tables show definitely how the rate increases as the interval between rupture and operation grows longer. Each hour after six increases the likelihood of general peritonitis.

In this series there were eight deaths, as shown in Table II.

TABLE II.

Showing Mortality in Thirty-Eight Cases.

Hours after Rupture.	Gastric Cases.		Duodenal Cases.	
	Number.	Deaths.	Number.	Deaths.
Under 12 ..	10	1	16	0
Over 12 ..	7	3	5	4

Several points of interest can be found on examining the fatal cases in detail. The only patient who died after operation within the twelve hours interval was a woman of sixty-one years with a large ulcer in the middle of the lesser curve. It was the only example of a large perforation, and eight hours after rupture the patient had general peritonitis with foul-smelling fluid and much œdema and inflammation of the peritoneum.

One man, forty-four years of age, came by ambulance from Nowra and was operated on sixteen hours after rupture of his ulcer. *Post mortem* examination revealed pneumonia, double empyema, subphrenic and pelvic abscesses.

One man, forty-eight years of age when rupture occurred, was rowing a boat at Sackville Reach, came to Windsor by motor car and then by train to Sydney and was operated on twenty hours after rupture.

One man, thirty-two years of age, had an ulcer which ruptured while he was driving a lorry at Botany. He drove it all the way to Homebush and was operated on twelve and a half hours after rupture. An interesting feature of this case was that he had had indigestion for

five years previously. An X ray examination four years before showed a gastric ulcer which was not found at operation in Balmain Hospital.

These three cases show how completely unfit for transportation the patients are.

A feeble old man of eighty-seven, with a history of more than twenty years' stomach trouble, was operated on seventeen hours after rupture. He showed not the slightest resistance and died of exhaustion rather than peritonitis.

The other three patients, aged fifty-nine, sixty-five and thirty-seven, were operated on respectively after twenty-four, twenty-four and fourteen hours. Four out of the eight patients who died were fifty-nine years old or more. This shows well the added seriousness in old people of ruptured ulcer.

There are some other points in the series worthy of interest. Drainage was not carried out, except when there was obvious gross soiling and inflammation, or when the fluid was offensive. Two tubes were used in most cases, one to the right kidney pouch through a stab wound in the right flank, and one to the pelvis by a stab wound above the pubes. Occasionally a third tube was inserted. Twenty-three of the thirty-eight abdomens were closed without drainage, and there were no deaths among the twenty-three patients.

Primary gastro-enterostomy was done in six cases with no deaths. I have not done it for five years. It has been more than once stated that perforation of a chronic ulcer and closure by suture is followed by healing. This is only partly true. Probably about 60% of patients are sufficiently well to carry on with their occupation. The remainder have recurrence of symptoms sufficiently severely to demand treatment, surgical or otherwise. I feel that it is better to wait for a recurrence of symptoms if they are going to occur, and then decide on the correct procedure after full investigation. Furthermore, gastro-enterostomy is not always the best operation for all ulcers. Primary partial gastrectomy was carried out in one case, as was primary excision. I believe these methods should be employed only when the indications are definite.

There was one case of subphrenic abscess following operation, which cleared up after being drained.

Another man developed all the signs and symptoms of subphrenic abscess. On X ray examination the diaphragm was twice reported as normal. Exploratory puncture of the chest under anæsthetic yielded no pus and, finally, a single intrahepatic abscess the size of a tennis ball was found in the inferior surface of the liver. This was drained and the patient recovered.

In three patients in the series rupture occurred twice.

The only other interesting case was that of a man, aged thirty-six years, who was involved in a drunken street brawl three days before admission to hospital and who was punched in the epigastrium, among other places. He had severe epigastric pain for three days, which became much worse fourteen hours before admission. He had a ruptured duodenal ulcer. This type of case suggests that violence may in some way lead to rupture of a chronic ulcer. The day following operation he got out of bed and attacked with a knife another patient, whom he accused of talking about him. For some days he had a noisy *delirium tremens* and was fed on rum and milk and went out of hospital quite well in the usual time; and alcohol is supposed to be a poison.

Reports of Cases.

TUBERCULAR TRACHÉO-ŒSOPHAGEAL FISTULA, WITH A REPORT OF CASE.

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THE fact that tracheo-œsophageal fistula is still comparatively uncommon and tuberculous fistulæ rare has prompted me to report this case with a short survey of the literature. It is probable that this rareness is due to the high mortality rate attending these cases and that fistulæ occur in patients with tuberculosis and cancer who die at home and never come into hospital. The rapidity with which death occurs is, however, not to be compared with that in the congenital cases, where it generally occurs in the first two weeks of life. Moersch[1] reports a case following carcinoma in a patient who lived eight years and then died of post-operative pneumonia. Moersch, in his survey of the reports of cases at the Mayo Clinic between 1916 and 1930, classified seventeen non-congenital cases as follows:

Carcinoma of œsophagus	9
Tuberculosis	2
Traction diverticulum	1
Actinomycosis	1
Dilatation of cicatricial stricture	1
Carcinoma in cicatricial stricture	1
Cause unknown (certainly not tuberculosis)	2
	17

The traction diverticulum case was probably also tuberculous, but in none of the three was a microscopic section obtained, the diagnosis being founded on a positive sputum in the two diagnosed cases.

Davidson and Mills,[2] in an article on the subject, find only two cases occurring in the *post mortem* reports of the Boston City Hospital for the previous twenty-five years—both carcinoma. There were twenty cases of carcinoma of the œsophagus which came to *post mortem* examination. Our own figures at the Adelaide Hospital for the past six years show that out of fifty-five carcinomata of the œsophagus only three cases of fistula were diagnosed. The carcinoma is practically always in the middle third, but most observations give a higher percentage of fistula occurrence.

The above authors mention as other causes: syphilis, as the result of two tertiary manifestations of the disease; aneurysm, as a result of erosion or pressure; inflammation, generally as a result of chemical agents or foreign bodies; and, rarely, œsophagomalacia, as an agonal manifestation in cases of cerebral disease in consequence of digestion of the œsophageal wall by gastric contents. Finally, they state that tuberculosis is described as a cause by Heddaeus[3] and Pehu.[4] They mention that, as well as the more direct form, it is possible for ulcerative processes in the lung itself to extend through the pleura to the œsophagus, and quote Pachnio[5] and Mikhailoff.[6]

Mangabeira Albernaz[7] found 639 cases in an exhaustive survey of the literature, but most of these were congenital or carcinomatous. There were twenty syphilitic cases. Factors of less importance were tuberculosis, actinomycosis and trauma, but the individual numbers are not given in the abstract I have seen. He reported one case following a gunshot wound.

The usual course is for the tuberculous disease to commence in the retrotracheal lymph gland. These, particularly the one in the neighbourhood of the bifurcation of the trachea, become attached to the œsophagus, with formation of a traction diverticulum, and then the caseous gland discharges into the œsophagus. This state was described as long ago as 1903 by Rivière.[8] Finally, the gland also discharges into the trachea and the fistula is complete.

Westergren and Ragnell[9] have reported two cases of this type: the first of a fistula connecting trachea and œsophagus, formed by the breaking down of a tuberculous mediastinal lymph gland; the second a case in which an œsophageal diverticulum had perforated into the right lower lobe, where extensive gangrenous processes were found.

It is possible for the disease to commence as a tuberculous ulcer of the trachea, but tuberculosis of the œsophagus is so rare that it can be left out of consideration. The only case I can find recorded is that described by Jackson in his "Œsophagoscopy and Bronchoscopy", 1927. Jackson[10] is also the only author I can find who mentions the diagnosis of a case having been established by removal of a piece of tissue for microscopic examination, so that the case I describe below must be quite rare.

Our own hospital reports show the following: During the last five years at Mareeba Babies' Hospital, Dr. Beare informs me that only two cases of the congenital type occurred; these were of the usual type, the lower segment of the œsophagus communicating with the trachea, while the upper segment ended in a blind pouch. At the Adelaide Hospital during the same period there have been only three carcinomatous cases. Another patient was admitted on August 20, 1924, under the care of Dr. de Crespigny, and in the presence of a strongly positive Wassermann reaction and no evidence of other disease the condition was diagnosed as syphilitic. No endoscopy was done in this case. Although no tuberculous cases other than the one I am describing have occurred, the following tuberculous lesions of the œsophagus have occurred in the *post mortem* room: two cases of traction diverticulum and in another instance a healed ulcer in association with an old healed tuberculous gland in a case of Pott's disease.

Case Report.

R.H. was admitted to the Adelaide Hospital under the care of Dr. Close, with the following history.

Four months ago, when having a drink, he had a sudden severe pain under the sternum and between the shoulders. Pain was not severe and was noticed only at times. A few weeks after the initial pain he started to bring up large quantities of whitish, slimy phlegm. At first he had no cough, but this soon developed, and persisted. When he took liquid food he got pain and fits of coughing, which made him vomit at times.

On examination the chest moved well on respiration. There was no impairment of percussion note. On auscultation there was a harshness of the breath sounds over the left side of the chest, anteriorly over the second, third and fifth interspaces. Posteriorly over the whole of the left side of the chest there was a grating sound heard and some loss of vesicular tone. X ray examination after a barium meal showed a small traction diverticulum of the œsophagus at the level of the bifurcation of the trachea. Some of the barium in the œsophagus was found to be escaping through this and down the left bronchus. This is shown by the accompanying illustration.

He was then transferred to me, and on May 10, 1932, under an anæsthetic, a bronchoscope was passed. A considerable amount of barium was still present, mainly in the left bronchus. At the level of the bifurcation a small ulcerated area bathed in mucopus was seen. After withdrawal of the bronchoscope an œsophagoscope was passed. The œsophagus was constricted at the level of the bifurcation and on the anterior wall there was an ulcerated area the size of a threepenny piece. A piece of this was removed with forceps for section and the report showed a tuberculous inflammation.

A method of examination which was not used in this case, but which has been suggested by Vinson, is to get the patient to swallow a thread. Vinson noted that in cases of a tracheo-œsophageal fistula the thread would find its way through the fistula and keep twisting itself around the tracheo-œsophageal wall by successively passing through the œsophagus after emerging from the larynx. Myerson[12] has reported a case in which this was the only evidence demonstrating the presence of a fistula.

The usual course of the disease is for the patient, after a short interval, to develop a bronchopneumonia as a result of the aspiration with the bronchi of the foodstuffs and other infected material from the œsophagus. In the tuberculous cases in which the fistulous communication between trachea and œsophagus is not direct, but generally by way of a broken-down tuberculous lymph gland, there is a chance that healing of the glandular process, even in part, may result in closure of at any rate a portion of the tract with relief of symptoms.

In the case I have described there seems lately to be some amelioration of symptoms, whether from the cause suggested above, I cannot say. At any rate the barium on the last examination did not pass into the trachea, although fluids still do sometimes at meals. Of course, the probabilities all point to an unfavourable termination. The patient can be fed, if necessary, through a stomach tube, which he is taught to pass himself. This was done in the syphilitic case mentioned above.

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(11) "Archives of the Adelaide Hospital", 1930.
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Reviews.

A TEXT BOOK OF ANÆSTHESIA.

"Practical Anæsthesia" is written by the anæsthetic staff of the Alfred Hospital, Melbourne.[1] It is the first of a series of monographs to be published by the Baker Institute of Medical Research. In the preface Dr. Penfold explains that the essential purpose of the series of monographs is to give an opportunity to the workers at the institute to write a succinct account of the subjects in which they have carried out research, so that they may incorporate their original work in the presentation, and from their personal experience may submit the work of others to a critical analysis. Similar monographs published by the Pasteur and Rockefeller Institutes have been of great value in medical education.

In recent years the practice of anæsthesia has become more and more specialized, while almost every medical journal describes some new method or new drug. It is impossible for the medical student to absorb more than "rag and bottle" methods in the short time devoted to the subject, while the general practitioner is unable to decide what may be of value in his work and what may be dangerous even in the hands of an expert. In this little book all this new work is reviewed simply, carefully and clearly, so that it is possible to ascertain the methods that are in the best interests of the patient. The illustrations are numerous and the value of each picture or diagram is enhanced by a very full accompanying description. In each section there is an illustration of the armamentarium required for that type of anæsthesia. These exhibits are well chosen with the exception of a few minor points, such as the Schimmelbusch mask, which is shown for ether anæsthesia, whereas that of Bellamy

[1] "Monographs of the Baker Institute of Medical Research: Number I: Practical Anæsthesia", by the Anæsthetic Staff of the Alfred Hospital, Melbourne; 1932. Australia: Australasian Medical Publishing Company, Limited. Demy 8vo., pp. 249, with illustrations. Price: 10s. 6d. net.

Gardner is preferable, as it is much more rigid and fits the face accurately. Figures XXIX and XXX show artificial respiration being performed on a patient whose head and shoulders are raised, although in practice the head of the table is usually lowered first. The index is full and accurate and each chapter has a list of references to original papers which may be consulted by those requiring further information.

The book shows the advantages and disadvantages of having a number of authors. Many of the chapters are clear-cut essays representing the latest work expounded by one who is a specialist in that department. In other chapters there is evidence that collaboration has produced a compromise between diametrically opposed views. However, the whole teaching is essentially practical and the patient's safety is stressed throughout.

The arrangement of the chapters is that in which the student should learn his work. Beginning with general considerations it passes on to the assessment of the anæsthetic risk, the choice of the anæsthetic, and preliminary medication before the individual anæsthetics and the methods of their application are discussed. The different sections are so uniformly good that it is difficult to select any for special mention. The chapter on the gaseous anæsthetics is particularly clear and in its seventeen pages gives a better review and more practical teaching than can be found in any of the larger text books. There are some, however, who will not agree that "the administration of the gaseous anæsthetics without oxygen, for brief anæsthesia, is now abandoned". The rôle of carbon dioxide in anæsthesia is of great importance and one of the major measures in reducing the number of fatalities. The reasons for its application and the methods of use are explained simply and clearly.

The chapter on spinal anæsthesia gives an unbiased summary of its indications and contraindications. The writer is wedded to "Ethocaine" ("Novocain") dissolved in cerebo-spinal fluid and injected in the sitting position. A little more discussion of the other methods would be an advantage, and also a description of the technique of injection in the lateral position. The routine premedication in spinal anæsthesia advised is one-quarter of a grain of morphine with one-hundredth of a grain of hyoscine. This dosage appears to be too high when considering the frequent use of this method in old people and in those whose renal function is impaired. It is also surprising to read: "Post-operative pneumonia after spinal anæsthesia is said to be markedly less frequent as compared with its incidence after inhalation anæsthesia". This is contrary to the findings of Sise, whose analysis showed that its incidence was equal in the two classes. Accidents and sequelæ of anæsthesia are considered very thoroughly. The section devoted to post-operative pulmonary complications is particularly good, the explanation of their mode of origin and proper treatment is comprehensive and concise. There is no mention, however, of the danger of exposing an anæsthetized patient in a cold operating theatre. Chloroform and its dangers are discussed with particular care, as would be expected in a book which is dedicated to Embley, whose researches in Australia were a great factor in establishing the proper place of chloroform. "Anæsthesia in Dentistry" is a most valuable chapter. It emphasizes the many advantages of the gas anæsthetics given continuously by the nasal and by the endotracheal methods. It is recommended that the anæsthetic be commenced with a mouth prop in position and the use of a mouth cover as well as the nasal inhaler. Induction, however, is easier and pleasanter without a mouth prop and using the nasal inhaler alone; the patient is asked to keep the mouth closed, the mouth cover should be reserved for mouth breathers and crying children. It is suggested that major dental operations should be performed in hospital; this excellent advice should be followed more frequently. Some experts will be unable to agree that "premedication is rarely practicable in ambulant patients". One worker with an experience of premedication in more than a thousand ambulant patients in both private and hospital practice has shown that it is of great benefit to the patient, anæsthetist and surgeon. Morphine may be given by hypodermic injection or the barbiturates, such as "Dial", "Allonal" or "Nembutal", by the mouth, without any fear that the patient will be too sleepy to go home.

In midwifery the various anæsthetics and narcotics are reviewed and their relative indications and limitations are discussed. The only omissions noted were chloroform capsules and the combination of "Nembutal" and chloral hydrate. A comprehensive summary is given of the present position of basal narcosis, which is described as "an effect greater than psychical sedation, and is associated with sleep which differs from the sleep of anæsthesia in that the patient may be aroused by painful stimuli". A warning is given that although basal narcosis is much appreciated by the patient, it may have disadvantages which are numerous and grave. Deep narcosis increases the risk of respiratory complications, and some inhalation or regional anæsthesia is usually required. An outline of regional and local anæsthesia is given that illustrates a technique which is suitable for all the operations usually performed by these methods. The manner of death during anæsthesia is discussed and a series of very sound recommendations are made which should help to lessen the number of these fatalities. Although this article is short, it is the best summary that has appeared in any small book on anæsthetics. The chapter on oxygen therapy is a valuable innovation and should enable anyone to master the essentials of this form of treatment.

This book is a credit to all those concerned in its production, and it shows a very high standard of work. It has already been chosen as a text book for students by at least one Australian university. The editor is to be congratulated on the general arrangement, the careful selection of illustrations, and the clear manner in which the information is displayed. "Practical Anæsthesia" may be confidently recommended to the student, house surgeon and practitioner as a guide to safe and scientific anæsthesia.

TREATMENT.

Dr. Robert Hutchison has recently published "The Elements of Medical Treatment".[1] It is deplored that Dr. Hutchison found it necessary to depart from his usual fund of details, for whilst undoubtedly there are elements of treatment, treatment cannot, or should not, be elementary. Throughout the work the author's long experience and valuable advice are seen to advantage in the discussion of principles, but in the detailed part of treatment there is much that is open to criticism.

This second edition of this work would have been of more value had the author taken into account the almost simultaneous publication of the British Pharmacopœia, 1932. "The number of drugs in the Pharmacopœia is very great", it is stated, "but most of them are superfluous." Yet as one reads one finds more than one drug recommended which has been deleted from the new pharmacopœia. Quite a number of proprietary drugs receive an undue mention and others of daily accepted use are neglected. The modern mass doses of digitalis in acute heart failure have not been considered. The tapping of an abdomen and the insertion of Southey's tubes are described, whereas such drugs as "Novasurol" or "Salyrgan" or even "Theocin", the use of which may obviate the necessity for either procedure, are not discussed. Surely the liver treatment of pernicious anæmia deserves more that the mere mention it receives? One statement, "Transfusion of whole blood is of great temporary value in all cases of severe anæmia", if acted upon, may lead to disaster in some cases of pernicious anæmia. In the chapter on treatment of diabetes the author enunciates much that is not in keeping with modern thought. The chapter on nephritis shows that when he speaks from experience one reads with interest, but the treatment outlined is open to criticism. Here in particular his "don'ts" carry weight.

After consideration we cannot recommend this book for the purpose for which it is written.

[1] "The Elements of Medical Treatment", by R. Hutchison, M.D., F.R.C.P.; Second Edition; 1932. Bristol: John Wright and Sons, Limited. Crown 8vo., pp. 188. Price: 5s. net.

The Medical Journal of Australia

SATURDAY, APRIL 29, 1933.

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A GREAT OCCASION.

THE year 1935 will be one of great importance to the members of the British Medical Association in Australia. As announced recently in the report of the last meeting of the Federal Committee, the Council of the parent Association has accepted the invitation of the Federal Committee and has determined to recommend to the Representative Body, which meets in July next, that the annual meeting of the Association for 1935 be held in Melbourne in September of that year. The Representative Body is very unlikely to fail to ratify the decision of the Council. The suggestion that the parent body might be asked to hold the annual meeting in Melbourne was made by a prominent member of the Victorian Branch; he thought that the meeting might be held during the centenary celebrations of the City of Melbourne, which will take place during the latter months of 1934 and the early months of 1935. Sir Henry Newland was asked to convey the invitation to the parent body. Unfortunately, the place of meeting for 1934 had already been determined; 1935 was then allotted to Melbourne.

The decision of the Home authorities will give the greatest satisfaction to the Australian Branches and doubtless also to the New Zealand Branch. One and all will congratulate the Victorian Branch on being chosen for the signal honour of acting as hosts to the Association. There is precedent for the holding of the annual meeting outside the United Kingdom. But recently the annual meeting was held in conjunction with the Canadian Medical Association, a body in affiliation with the British Medical Association. Never before, however, have officials of the Association journeyed so far in a body. (We are given to understand that possibly about one hundred members will make the journey from the old country.) The significance of the meeting will be great. A mother will be visiting her children, children whom she has scarcely seen since she sent them out into the world. She will find them grown almost beyond recognition and some of them straining at the leash for still greater freedom. The children, on the other hand, will find a mother who knows all about them, who has their interests at heart, a mother whose influence is far more widespread and potent for good than they ever imagined. It is not too much to say that the meeting will be mutually beneficial. While, on the one hand, the mother will deepen her understanding and will try more than ever to meet the needs of her children, the children will doubtless turn a loyalty born of tradition into a loyalty based on admiration and affection. Needless to say, the children will derive the greater benefit.

The Melbourne meeting will, as is usual with annual meetings of the Association, be devoted to sectional discussions on the science and practice of medicine. It will have two effects. It will show to the general public that the British Medical Association is not, as many regard it, a trade union, concerned mainly with money and the personal advantage of its members. The public will see medical practitioners coming together from all parts of the Empire to discuss the causation of disease, its prevention and its cure; they will realize that scientific knowledge and medical equipment are the main thought of the members of the Association. The members of the Australian Branches will meet

in what is virtually a British Medical Association congress.

This at once opens up an avenue for future development. There is no reason, so it appears to us, why a meeting analogous to the annual meeting of the Association, but held, say, every two years, should not eventually take the place of the Australasian Medical Congress (British Medical Association). Such a gathering could be held in each State in turn and would be open to members of the Association without payment of a special congress fee. Special arrangements would have to be made by the Federal Council for the financing of such gatherings and the participation of New Zealand in any such arrangement should be secured. There would, of course, be difficulties in carrying out such a proposal and possibly objections, but the suggestion is made for the consideration of the Branches.

Current Comment.

LEPROSY.

Opinions on the necessary measures of controlling leprosy have undergone a gradual change during the past few years. In 1926 Molesworth advocated the abolition of segregation; but his strong plea for a more humane attitude towards lepers was at the time unheeded. Molesworth's views on leprosy are well known, and there is no need to repeat them here; suffice it to state that his main argument is to the effect that the incidence of leprosy depends on individual and racial resistance and that the disease dies out or diminishes in a community through the development of this resistance, or rather, the survival and increase of a resistant racial stock. Authorities in various parts of the world, whether they accept Molesworth's views or not, are now coming to recognize the futility of any hide-bound rules of segregation. Improvements in treatment, of course, have a good deal to do with the changed ideas; it is now possible in many cases to promise a cure. The advocates of segregation were formerly influenced by the knowledge that little or nothing could be done in the way of treatment, and that any patient at large would inevitably become more extensively diseased and a greater source of danger to the rest of the community. It is remarkable that for many years after the discovery of *Bacillus lepræ* so little thought was given to individual infectivity and individual resistance. This aspect of the problem was considered by Muir, who, in 1929, advocated efficient treatment at clinics of all non-infective lepers and segregation of the infective only. Muir has had many years' experience in leprosy in India and must be regarded as a leading authority. His views in 1929 were vastly different from those expressed in a book written by himself and Rogers a few years earlier. In a recent paper dealing with the relief of leprosy in India he lays great stress on the necessity of careful survey work and the institution of clinics and gives scant space to segregation.[1]

He mentions that industrial enterprise has a good deal to do with the incidence of leprosy in India. Workers in some industrial areas are drawn from all parts of the country and are of different classes; they tend to discard their class rules when they are away from home, and they intermingle, often in insanitary and crowded conditions. It has been found recently that the incidence of leprosy is at least 1% in nearly all industrial areas, and as high as 3% in some. Muir remarks that a heavy responsibility rests on the persons concerned with the sanitation of these places.

He goes on to discuss the policy of the British Empire Leprosy Relief Association. The aims of this Association are: early diagnosis, early treatment, and prevention. In an endeavour to attain these ideals, the Association makes provision for the training of medical practitioners in the recognition and treatment of leprosy, arranges leprosy surveys, employs propaganda concerning leprosy, and encourages research. Muir points out that the study of leprosy has been neglected by medical practitioners because until recently the disease was regarded as incurable; consequently many medical practitioners are unable to diagnose any but the obvious cases. Instruction concerning leprosy is now given in many medical schools in India, and short courses for medical graduates are conducted at Calcutta several times a year. By these means and by the distribution of suitable literature it is hoped that within a few years most of the medical practitioners in India will be as well informed on leprosy as they are on malaria. A leprosy survey of large and thickly populated areas is a gigantic task. It has been found that five skilled workers take a month to survey an area with a population of 40,000; it would take seventy such parties ten years to survey the whole of India. The British Empire Leprosy Relief Association therefore appointed a party which visited numerous provinces and states, giving demonstrations and endeavouring to induce each province or state to appoint its own survey parties to work along similar lines. Treatment is carried out as part of the survey. A clinic is set up in a highly endemic area. Contacts of patients suffering from leprosy in an infectious stage are examined in their villages. The people are shown, by various means, the early signs of the disease and are taught how infection is spread. Their good will and confidence are won and, instead of hiding the disease, the sufferers come forward in the hope that they may be cured. Muir remarks that a much more thorough census can be taken by

[1] *The Indian Medical Gazette*, February, 1933.

means of a method including treatment and propaganda than by any other means.

Among the interesting pieces of information brought to light by the survey work are the following: (i) Leprosy is most prevalent among tribes who have left their native seclusion, but have not yet adopted modern sanitary measures. (ii) The commonest form of leprosy is nerve leprosy, which is presumably non-infectious; but in some cases the neural forms progress to the infectious cutaneous type. (iii) The finding of large numbers of slight cases in a village indicates either that the disease has been newly introduced and is spreading rapidly, or that it has been long present, but occurs in a mild form on account of the high resistance of the inhabitants. (iv) There is generally a more or less obvious predisposing cause, such as some other disease or malnutrition; causes vary with localities and individuals.

As a result of the British Empire Leprosy Relief Association's activities great numbers of special leprosy clinics have been established and provision has been made at many general dispensaries for the treatment of leprosy on special days; it has become generally recognized that the leprosy problem, though difficult, is not insoluble.

Muir stresses the necessity of instructing natives concerning the mode of spread of leprosy, in particular of warning them of the danger of exposing children to infection. The object is to encourage them to institute their own systems of isolation of the infective. In native villages sick and healthy members of a family sleep together in a small crowded room in conditions that must be nearly optimum for the transmission of leprosy. This particularly is an evil the villagers must be educated to avoid. Infective lepers might be permitted to move about freely at their work in company with others, but should have separate sleeping accommodation and separate eating and drinking utensils. It is impracticable to isolate all infective lepers in institutions in India. Muir goes on to state that the great predisposing causes of leprosy in India are the bad social and hygienic conditions of the people, and that all measures tending to improve these conditions will tend to bring about a diminution in the incidence of leprosy. "Leprosy propaganda should therefore merge into the wider sphere of general public health work."

Muir's views are logical and the methods he advocates are sound in the light of present knowledge. No other method would be likely to be of much value in the Territory of New Guinea, where there is a great deal of leprosy in some communities. Melanesians are ignorant, dirty and superstitious, and have a dread of isolation, often a dread of admission to hospital. Any method providing for isolation in institutions and neglecting the native village life would be doomed to failure; any attempt at a survey would be hopeless unless the natives' confidence was first obtained, and confidence could not be obtained while there was any threat of segregation. It will be interesting to see whether the experience of the next three or four years will still further alter Muir's opinion.

In the course of time, perhaps, leprosy will die out or become of minor importance in India. Will Molesworth's views then be accepted (they will not be disproved), or will the change in conditions be ascribed entirely to anti-leprosy activities? Possibly by that time there will be sufficient enlightenment to make the reason obvious.

INTESTINAL AMŒBIASIS.

The treatment of intestinal amœbiasis is unsatisfactory. True, acute attacks can be controlled by the injection of emetine; but in the majority of cases the infection remains after the acute dysentery has subsided. Various drugs, particularly emetine bismuthous iodide and emetine periodide, have been employed with the object of destroying the infection; in many cases all fail. It has been shown within recent years that the bark of *Holarrhena antidysenterica* (known in India as *kurchi*) possesses amœbicidal properties. Alkaloids known as conessine, kurchine and kurchicine have been isolated. In 1929 H. W. Acton and R. N. Chopra showed that these substances could be used in the treatment of human amœbic dysentery; recently they have published another report on the subject.[1] They state that intramuscular injections of the total alkaloids are as effective in the treatment of acute attacks as emetine, but that they cause severe irritation and pain at the site of the injection. These alkaloids have no toxic effects, and as soon as a non-irritant salt can be prepared, they will no doubt be widely used in the treatment of acute amœbiasis. In the treatment of chronic amœbiasis, a bismuthous iodide compound of the total alkaloids, termed "kurchi bismuthous iodide", is administered by mouth in a dose of 0·6 gramme twice a day for ten to twenty days. As the intestinal contents are apt to be strongly acid, and the action of the alkaloids is weakened by acidity, an alkali is given half an hour before each dose. The proportion of probable cures to failures noted by Acton and Chopra when this treatment was used, was 3·16 to 1, as compared with 1 to 3·5 noted by Knowles in similar cases when emetine bismuthous iodide was employed. The criterion of cure was the inability to find *Entamœba histolytica* in the stools on five or more different days after the cessation of treatment. Kurchi bismuthous iodide may be given concurrently or alternately with the seeds of *Plantago ovata* (known in India as *isabgul*). The mucilage from these seeds forms a protective covering to the amœbic ulcers, inhibits bacterial multiplication and absorbs bacterial toxins.

Kurchi bismuthous iodide may be the drug for which thousands of sufferers from chronic amœbiasis have been praying. But it must not be forgotten that the advent of other drugs has been hailed just as joyfully. Its non-toxicity is an important feature in its favour.

[1] *The Indian Medical Gazette*, January, 1933.

Abstracts from Current Medical Literature.

MEDICINE.

The Prediabetic State.

J. D. TYNER (*Clifton Medical Bulletin*, July-October, 1932) discusses the incidence of the prediabetic state as determined by the Brill test meal. This meal consists of 100 grammes of carbohydrate, 25 grammes of protein, 25 grammes of fat, a total of 725 calories. Blood is taken from a vein just before and again two hours after the meal, and the blood sugar is estimated. The urine is tested just before and again four hours after the meal. The Benedict copper method of blood sugar estimation is used. The result of the test is normal if the postprandial blood sugar is below 100 milligrammes per 100 cubic centimetres of blood; above this figure the patient is in the prediabetic state, and a blood sugar content above 150 milligrammes per 100 cubic centimetres indicates true diabetes. Reports on 1,500 patients with arthritis, hyperthyreoidism, hypertension and other conditions suspected of being associated with a prediabetic state, are given. Of obese patients, 51% were prediabetic, whereas only 44% of thin subjects were so classified. Prediabetes is more frequent in hyperthyreoid patients, in those with arthritis, hypertension, chronic cholecystitis with stone, old age and arteriosclerosis.

The Consumption of Carbohydrates in Relation to Disease.

J. H. P. PATON (*Edinburgh Medical Journal*, September, 1932) draws attention to the increasing and excessive consumption of carbohydrates, which is largely due to the additional use of cane sugar in the dietary. With the increased consumption of cane sugar there has been no concomitant reduction in the intake of cereals. To this excess the author attributes the high rate of incidence of catarrhal affections in children, the frequency of acidosis, rickets and dental caries and the increasing death rate from cancer and diabetes. The amount consumed per head *per annum* varies from 112 pounds in Australia, New Zealand and the United States of America to 65 pounds in Austria. To obtain 100 pounds of sugar annually from natural sources each individual would have to consume more than two pounds of sugar cane or beet daily. Though it has been commended as a food for children, sugar, as manufactured, is neither a natural nor a necessary food, since it is a pure chemical substance, which occurs only in a few plants and in dilute form. Most of the sugar consumed is surplus and throws an undue strain on the liver and pancreas. Because it is cheap it comes to replace other foods and there is a resulting deficiency especially of fats and vitamins. The lævulose derived from the digestion of su :ose is of doubtful value, and in large amounts is possibly toxic. When five grammes of sucrose per kilogram of body weight are eaten by a healthy individual sucrose appears in the urine and continues to appear for some hours. The inverting power of the gastro-intestinal juices is not unlimited. Continuous ingestion of excess of starch or sugar may be tolerated for a time, but eventually progressive failure of the digestive powers may be expected. Absorption of glucose beyond an optimum amount increases the water content of the body, which is the primary fault in catarrhal conditions. The observed reduction of catarrhal illness in a boarding school during the war years was associated with the rationing of sugar. It may be concluded that prolonged ingestion of excess of carbohydrate will result in hyperinsulinism and finally in hypertrophy of the pancreas. If atrophy succeeds hypertrophy permanent diabetes will follow. Where hyperinsulinism is present, an attack of febrile illness, through inhibition of digestion and absorption, will be followed by a comparative hypoglycæmia with the rapid formation of diacetic and oxybutyric acids. This may explain the beneficial effect of temporary glucose feeding on children with acidosis. Experimental work by Mellanby and Lee Pattison indicates that mere excess of carbohydrate in the diet appears to inhibit the activity of vitamin *D*.

Comparative Efficiency of Staining Methods for Tubercle Bacilli.

C. J. KOERTH AND R. J. B. HIBBARD (*The Journal of Laboratory and Clinical Medicine*, February, 1933) have found the Spengler method of staining for tubercle bacilli to be four times as efficient as the Ziehl-Neelsen method. Their conclusion is based on the result of the examination of 18,000 fields on 1,800 slides made from the sputa of 600 patients. It is recognized that at various stages of growth the tubercle bacillus varies in its acid-fast quality. Some are not acid-fast, some are weakly acid-fast, and some are strongly staining acid-fast. For some years the writers have used the Ziehl-Neelsen, the Schulte-Tigges and the Spengler methods in every specimen of sputum examined. Their technique for each stain is given. In using the Spengler method they stain with carbol-fuchsin (warm, but not hot enough to cause bubbling) for five minutes. The stain is poured off and without washing picric acid alcohol (equal parts saturated aqueous solution of picric acid and 95% alcohol) is poured on for four to five seconds; the slide is washed with 60% alcohol and treated with 15% nitric acid till a yellow colour appears (not over 30 seconds); it is then counter stained with picric acid alcohol solution until the colour is lemon yellow; it is washed with distilled water, dried and examined. Bacilli are brownish-red against a yellow background. The Schulte-Tigges method is but slightly less efficient than the Spengler. It is much more difficult to perform and the chance of error in technique is large. The Ziehl-Neelsen technique has no advantage over the Spengler in respect to the time-saving element and, if acid alcohol is used, is actually much more tedious. The Spengler method, as described, takes approximately seven minutes.

Thyreoid Gland Deficiency in Chronic Arthritis.

F. C. HALL AND R. T. MONROE (*The Journal of Laboratory and Clinical Medicine*, February, 1933) have analysed 300 cases of chronic arthritis for signs and symptoms of thyreoid deficiency. By chance 150 were of the atrophic (rheumatoid) type and 150 of the hypertrophic (osteo-arthritic) type. Where a case was found to be of mixed type it was placed in the group to which it most nearly corresponded. The diagnosis was arrived at through the gross appearance of the joints and the X ray findings. In addition to noting the commonly accepted clinical signs and symptoms of hypothyreoidism the authors determined the basal metabolic rate in a large proportion of the patients and the effect of thyreoid gland therapy was observed in those patients in whom its use seemed to be indicated. In the hypertrophic group the signs and symptoms of thyreoid inadequacy were common, the basal metabolic rates were below −10 in 50% and below −15 in 34·2% of 108 patients (in 42 patients no determinations of the metabolic rate were made), and thyreoid gland therapy was of permanent beneficial effect in 49·1% of 116 patients in whom its use seemed to be called for. In the atrophic group of patients the evidence of hypothyreoidism was less frequently noted and the signs and symptoms suggesting it could generally be ascribed to depletion and undernutrition. Basal metabolic rates, determined in 106 patients, were below −10 in 35·6% and below −15 in 17·7%, while thyreoid gland therapy was of permanent beneficial effect in only 16·5% of the 103 patients on whom it appeared worth trying. The writers believe that in certain patients suffering from non-tuberculous chronic arthritis, particularly where it is of the hypertrophic type, thyreoid deficiency, in addition to infection, dietary deficiency, trauma and depletion, may be a contributing factor in the ætiology, and that its correction, if present, affords relief of symptoms and aids in arresting the condition, probably through better joint nutrition.

The Treatment of Certain Cardiac Arrhythmias with Potassium Salts.

J. L. SAMPSON AND E. M. ANDERSON (*The Journal of the American Medical Association*, December, 1932) describe a further series of experiments on the effect of the administration of potassium salts to patients suffering from certain cardiac arrhythmias. They claim that prior to their pre-

liminary report in November, 1930, potassium had not been used to control the heart rhythm in man. Potassium salts were given by mouth to 58 patients known to have auricular or ventricular ectopic beats or paroxysmal auricular or ventricular tachycardia for an appreciable period of time. One of the patients received in addition ten cubic centimetres of a 1% solution of potassium chloride intravenously without ill effect, but with no demonstrable advantage over the oral route of administration. The chloride, bromide, iodide, citrate and acetate salts, when applied to the same patient, had apparently identical effects. The acetate caused less gastric distress than any of the other salts, and was adopted for routine use in a 25% aqueous solution. Among the 58 patients complete eradication of the arrhythmia was obtained in 29; of these two had demonstrated ventricular ectopic beats accompanying an overdosage of digitalis. The remaining 29 did not show a definite favourable response, though six had an apparent diminution of the number of ectopic beats. Three showed what were probably paradoxical effects, one initiating paroxysmal ventricular tachycardia during the administration of the potassium and developing severe somatic muscular cramps, which disappeared on its withdrawal, and the other two showing apparent increases in the number of ventricular ectopic beats. The average age of patients showing favourable response was sixty-one years and of those showing no response forty years. At first doses of from one to four grammes were tried, but so inconstant were the responses as to suggest lack of correlation between the size of the dose and the therapeutic effect. So great were the variations from unknown sources that the patient's weight was not taken into account in calculating the dose. The threshold of potassium influence on heart arrhythmias varies widely and no law can be discovered at present that governs this, other than possible variations with age. In the case of a woman, aged forty, with showers of ventricular ectopic beats, but without organic heart disease, initial doses effective in other cases, six and twelve grammes, were below her threshold, and only when sixteen grammes had been administered did her arrhythmia disappear. The use of subeffective doses may account for many of the failures. In the majority of cases response occurred in about forty minutes, with a minimum of twenty and a maximum of ninety minutes, and varying somewhat with the dose. The duration of effect also varied with the dosage. From three to eight hours was the range for most cases. Other than the effects ascribed to paradoxical action there were no serious clinical disturbances caused by the doses of potassium salts given. Contrary to anticipation, diuresis was uncommon. Increased age and the presence of organic heart disease seemed to lower the threshold of demonstrable effect of potassium salts on auricular and ventricular ectopic beats and tachycardia. The theory of the mode of action is discussed.

Blood Sugar and Glycosuria.

ADLERSBERG AND PORGES (*Deutsche Medizinische Wochenschrift*, October 7, 1932) discuss the relationship between blood sugar and glycosuria. They conclude: (i) True diabetics with a low renal threshold respond to diet and insulin, their insulin-glucose equivalent is high and they are very susceptible to insulin, and with insulin therapy it is difficult to make them aglycosuric without provoking hypoglycæmia. (ii) True cases of diabetes occur with glycosuria and normoglycæmia. In these cases one cannot produce aglycosuria without producing hypoglycæmic attacks. (iii) Cases of normoglycæmic glycosuria (so-called renal glycosuria) without diabetic metabolic disturbances occur. These patients cannot be made aglycosuric, otherwise hypoglycæmia occurs and they are susceptible to insulin, for it lowers their blood sugar. To differentiate them from (ii) a glucose tolerance test shows only a very slight increase in their glycosuria, and the same happens with a rich carbohydrate diet. Cases of renal and diabetic glycosuria must be distinguished, the former being harmless; the latter, though mild, will progress. (iv) The glycosuria of diabetics with a high renal threshold responds sluggishly to diet and insulin. These patients show a low insulin-glucose equivalent and very often at the start manifest little or no response to insulin.

Ovarian Hormone Therapy in Ovarian Insufficiency and the Menopause.

B. WEBSTER (*The American Journal of the Medical Sciences*, December, 1932) reports the results of the administration of ovarian follicle hormone therapy to twenty patients suffering from conditions commonly ascribed to ovarian insufficiency. Two types of patient were selected for treatment, ten were at the menopause, of which two were artificial and eight were natural, and ten suffered from primary amenorrhœa. The climacteric patients were all selected because of the severity of symptoms such as hot flushes, pruritus, headache and nervousness. Cases with complicating hypertension were not used. In the ten cases of amenorrhœa there was considerable variation as to type, but all were shown to be free from anatomical lesions of the genital tract. This group could be divided into two types. The one was regarded as simple ovarian follicle hormone insufficiency and the other as belonging to the anterior pituitary ovarian dysfunction syndrome. The ovarian follicle hormone used was prepared according to Morrell's modification of the Allen-Doisy technique and was administered by means of vaginal pessaries. The dosage appears to require adjustment with each individual case. No medication other than the specific hormone was given. During the control period pessaries of plain gelatine were used without the patient being informed. Strikingly beneficial results were obtained in three of the eight cases of natural menopause. In each of these three cases the menopause was of less than one year's duration. There was marked relief of symptoms and reestablishment of uterine bleeding. In three of the five cases in which the menopause was of two to five years' duration there was considerable symptomatic relief, but no return of menstrual bleeding. The two patients with artificial menopause both obtained remarkable relief from headaches, hot flushes and nervousness, but not until nearly 1,000 units had been used. The relief continued for approximately one week after therapy was stopped during a control period. In one patient marked congestion of the breasts was noted after the use of relatively large quantities of the hormone. The two patients with artificial menopause could be maintained symptom-free on approximately 500 units per month. No definite effect on the blood pressure was noted in any of the cases. There was a loss of ten pounds or more in weight in the course of the three months' duration of the experiment in six of the ten cases. The cases of amenorrhœa which were regarded as being due to simple follicle hormone insufficiency gave a good response. No beneficial results were obtained when the hormone was administered to patients with amenorrhœa who showed clinical evidences of an anterior pituitary dysfunction.

The Prognosis in Diabetic Coma.

BERTRAM (*Münchener Medizinische Wochenschrift*, October 7, 1932) states that the prognosis in diabetic coma can be given by simple methods. The blood pressure should be estimated regularly. A blood pressure below 100 millimetres of mercury in a hypertomic diabetic renders prognosis doubtful. A continuous fall of blood pressure in spite of cardiac stimulants and suitable coma treatment renders prognosis almost hopeless, even should the person awake from his coma. In an asthenic diabetic the blood pressure at the onset is not so important, but prognosis is grave when the blood pressure keeps on falling during the first eight to ten hours in spite of all treatment. Most of these patients die of cardiovascular failure. Prognosis is extremely bad and cannot be determined when the coma has lasted more than eight hours. In uncomplicated cases prognosis is good when tendon reflexes are still present. The author describes the cause of coma under three headings: (*a*) Acute infections; prognosis here depends on ætiology and the factors already mentioned. (*b*) Myocardial and renal disease in hypertonic diabetes; here prognosis is bad. (*c*) Gastric and colonic disease with dehydration; here prognosis is bad. Apart from high values, the blood sugar concentration is unimportant. Prognosis is very grave with a blood sugar of 0·7% to 0·8%.

Special Articles on Treatment.

(Contributed by Request.)

VII.

THE TREATMENT OF RETROVERSION OF THE GRAVID UTERUS.

THE non-recognition or ineffective treatment of retroversion of the gravid uterus may cost a woman her life or a doctor his reputation.

Neither this nor any other pathological condition can be successfully dealt with unless a correct diagnosis has been made; therefore one must have a clear conception of the means by which this may be attained.

The patient will probably seek advice for frequent or difficult micturition and pelvic discomfort associated with a missed period or periods and morning sickness. The physician will note breast changes, a deep-seated purple discoloration of the vaginal mucosa, the os anterior and a globular elastic mass in the pelvis. He must not hastily conclude that this is the gravid uterus and thereupon attempt to push it up. I have seen disastrous consequences follow from mistaking a hæmatoma of the broad ligament due to rupture downwards of a tubal pregnancy for a gravid uterus.

A tubal pregnancy which has ruptured into Douglas's pouch and formed a hæmatocele there, or a small ovarian cyst in Douglas's pouch may also simulate a gravid uterus. It is evident that the treatment which would be correct for the latter would be in the highest degree mischievous for the other conditions mentioned. To avoid such serious errors it is only necessary to bear in mind their possibility and exclude them by a painstaking bimanual examination. Although the subject of diagnosis is beyond the scope of this article, it must be stated that the attending practitioner should note that the side borders of the cervix can be traced by the finger downwards and backwards to merge into the body of the retroverted gravid uterus, while when an extrauterine mass is present in the pelvis, pushing upwards and forwards the cervix towards the pubic arch, the uterine body can be detected perched up above the pubis or perhaps flattened out on the tumour mass. The meatus is displaced upward in the incarcerated uterus, from traction upwards of the anterior vaginal wall, constituting another important point in the differential diagnosis.

If untreated, a retroversion of the gravid uterus may rectify itself, but this is extremely unlikely. The usual course is for an abortion to occur. In my experience this condition is the most common cause of abortion and could be easily eliminated by medical supervision from the beginning of pregnancy. Sometimes the pregnancy continues to develop until the uterus fills the pelvis, causing serious pressure symptoms and retention of urine. The blood supply of the bladder may be so interfered with that the entire mucosa sloughs away. An example of this was shown by the late Dr. Newmarch at a Branch meeting.

Once a careful examination and a correct diagnosis have been made treatment should be prompt. The first step, and that an essential one, is to empty the bladder. If there has been retention of urine, this emptying should be very gradual. Not more than ten to twelve ounces should be drawn off at one time. One should wait half an hour before evacuating more, or a very fine catheter, if available, could be used, which would restrict the flow to a mere trickle. Much has been recently written on "gradual decompression" and on the great risk of rapid relief of tension, which in the bladder might cause such a rebound as to increase greatly the risk of sloughing of the mucosa.

The bladder having been emptied, an attempt is made to restore the uterus bimanually, pushing the fundus upwards with two fingers as high as possible and then suddenly transferring the forefinger anteriorly and endeavouring to draw the cervix backwards and upwards while the external hand above the pubes endeavours to take charge of the elevated fundus and to coax it forwards.

Owing to the loss of the natural rigidity of the uterus, due to pregnancy and malposition œdema, this method will often fail in the absence of anæsthesia. We then resort to the second method, restoration in the knee-chest position.

The patient's thighs should be at right angles to her body and the lumbar region well hollowed. Two fingers of the left hand are then passed into the vagina and the perineum should be drawn strongly upwards, admitting atmospheric pressure. In order to gain advantage from this atmospheric force, it is necessary to seize the anterior lip of the cervix with a traction forceps and to roll it backwards while at the same time the two fingers of the left hand push the body of the uterus forwards and upwards, avoiding the sacral promontory.

The patient is then turned into the dorsal position and a careful bimanual examination is made to ascertain if the uterus has been completely restored. It is wise to insert an Albert Smith vulcanite pessary and to allow it to remain for two or three weeks, that is, until the uterus has become too large to become retroverted again.

Adhesions may be present which make it impossible to effect restitution of the uterus. In these circumstances the best treatment is to open the abdomen, gently separate all adhesions, and lift the uterus out of the pelvis.

Aspiration of the *liquor amnii* by a puncture through the posterior vaginal wall and posterior wall of the uterus, after which the uterus can be restored bimanually, has been done, but, in so much as such a procedure must inevitably lead to abortion, it should be avoided if possible.

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British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SECTION OF NEUROLOGY AND PSYCHIATRY OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the Repatriation Division of Callan Park Mental Hospital at 2.15 p.m. on Thursday, December 1, 1932, DR. A. W. CAMPBELL in the chair. The meeting took the form of a series of clinical demonstrations.

Psychosis Associated with Paget's Disease.

DR. J. M. RAINBOW showed a patient who was suffering from psychosis associated with *osteitis deformans*. The patient had been shown at a previous meeting and was shown again so that his progress might be noted. The patient was a man, aged thirty-nine years, who began to show signs of mental illness in 1927. He then complained that his nerves were bad and that "all sorts of subjects ran through" his mind. He began to lose interest, suffered from insomnia and felt generally unwell. He expressed unusual religious ideas and believed he was "doped".

He was admitted to Callan Park Mental Hospital on November 24, 1927. He was physically a tall, thin man, with a noticeable bulging of the right fronto-parietal area. Superficial temporal arteries were tortuous and pulsated visibly. All his movements were slow, and his eyelids tended to droop. His mental condition was one of stuporose confusion. The bony deformity gradually increased, and in 1930 the cranium was decidedly larger and he exhibited kyphosis and slight anterior bowing of the long bones. Cranial measurements were as follows: Circumference, 57·1 centimetres (twenty-two and seven-eighths inches); over the prominence, 58·75 centimetres (twenty-three and a half inches); vertical, through the external auditory meatus, 36·25 centimetres (fourteen and a half inches); and nasion to occipital protuberance, 39·6 centimetres (fifteen and seven-eighths inches). The X ray report stated that well marked Paget's disease of the lumbar spine and skull was present. His physical condition further progressed, and in 1932 the cranial measurements were as follows: Circumference, 58·4 centimetres (twenty-

three and three-eighths inches); over prominence, 60·0 centimetres (twenty-four inches); vertical, through the external auditory meatus, 37·5 centimetres (fifteen inches); and nasion to occipital protuberance, 39·6 centimetres (fifteen and seven-eighths inches). There was also dorso-cervical kyphosis, spreading of the base of the thorax, slight anterior bowing of tibiæ and femora.

Although his mental state for years had been one of dull, uncommunicative, inaccessible, faulty, apparently demented state, there was a surprising improvement during the later months of 1932. It was then noticed that he conversed freely and coherently and was oriented correctly in all spheres. He could give a good account of himself, was clean in habits, and generally much improved. However, he was somewhat childish and inert, and talked in a rambling way of "visions of different individuals". The marked change in his mental state showed only mild dementia present and seemed to remove any possibilities of damage to the brain by direct bony pressure. There were also no localizing signs or symptoms of any sort indicative of brain involvement. His physical condition had progressed further than his mental deterioration and seemed merely coincident and not causal.

Case for Diagnosis.

Dr. Rainbow also showed a case for diagnosis (psychosis with organic disease of the central nervous system). The patient was a male, aged forty-nine years, born in New South Wales, a draughtsman. The present illness was of about two years' duration. He began to be troubled with his walking; it progressed and, finally, about twelve months ago, the patient was superannuated. He lived at home with relatives, but during the last six months he had become restless, irritable, dirty and objectionable, and generally hard to manage. The family history showed nothing unusual. He was always healthy and all his life worked in a government department. He married at the age of twenty-five, but was divorced six months later. A sister stated that all through his life he had been bad-tempered and at times irritable and quarrelsome. He was a non-drinker, but a heavy pipe smoker. He denied the risk of venereal disease.

He was admitted to Callan Park Mental Hospital on October 27, 1932. He was certified as being slow and almost incoherent in speech, foolish in antics and mannerisms, unduly cheerful, rambling and contradictory in statements, mistaking identities, and with no insight. On admission to Callan Park he was unduly cheerful, with no real appreciation of his condition. He was not able to give a strictly reliable account of himself or his illness, being contradictory and inclined to ramble. He said he had been at Callan Park for weeks and it was September, 1932. However, he knew where he was and could give his home address correctly. His remote memory was good, but his recent memory was not so good. He was slow and vague in thought and generally there was some degree of cerebral deterioration. Physically he was a tall, thin, middle-aged man, obviously "fidgety", and displayed sudden involuntary movements of head, neck, upper limbs and occasionally of the trunk, in all directions. There were also champing movements of the jaws, frequent pursings of the mouth and frequent sudden grunts. These movements, especially of the right extremity, were choreiform and at times athetoid. The movements were increased when the patient was disturbed. He could not stand for long without support, his balance being very precarious; and he walked with great difficulty on a wide base and with the body bent forwards. This apparently was due to weakness and rigidity of his lower limbs.

There was no loss of smell. His vision was apparently unimpaired; he could move his eyes in all directions, but slowly, and cooperated badly. No nystagmus was present. His face was somewhat inexpressive, there was doubtful rigidity of facial muscles, but no paresis. He appeared to have difficulty in initiating facial movements, for example, he screwed up his eyes after a long interval; he also appeared to have difficulty in opening his mouth. The tongue on protrusion deviated to the right. No deafness or other abnormality of the eighth nerve was noticed. Taste was unimpaired. There was no loss of swallowing power. Speech was indistinct and slurring. All movements were clumsy, due to generalized hypertonicity and paresis of muscles, for example, both upper and lower extremities were somewhat spastic and paretic. There was no marked incoordination. The rigidity of the right lower extremity seemed slightly greater than the left. Hypertonicity seemed more "clasp knife" in nature than "cog-wheel". Slightly more coordination was present in the lower extremities. There was no wasting of muscles. He was able to appreciate all forms of gross stimuli. Passive position and passive movement were unimpaired, but cortical discrimination was impaired. Cortical localization was practically unimpaired. All reflexes were present and exaggerated. The plantar reflex was said to have been extensor at the time of his admission to hospital; a few weeks later it was indefinite and if anything seemed flexor. Ankle clonus was present on the right side; no patellar clonus was present on either side. The pupils were of moderate size, of regular outline, and reacted to light and accommodation and consensually. The Wassermann test gave no reaction with either blood or cerebro-spinal fluid. The fundi appeared physiological. There were no trophic disturbances. The circulatory, respiratory and alimentary systems were clear, but there was pyorrhœa with several carious discoloured teeth and stumps.

A few weeks after his admission his involuntary movements were much less evident. However, he remained rigid and clumsy, with the other signs found on admission still present. There was occasional loss of control of bladder and rectum. Mentally he remained somewhat euphoric, though at times irritable, and a general cerebral deterioration persisted.

Because of his cerebral deterioration, impaired speech, choreiform movements and loss of control of his lower limbs he appeared at first something like a patient with Huntington's chorea, but the absence of family history and subsidence of the movements excluded this possibility. Tabo-paresis was eliminated by the presence of the deep tendon reflexes, by the fact that joint or muscle sense was not lost; by hypertonicity, lack of eye signs and of trophic changes; also by the failure of blood and cerebro-spinal fluid to react to the Wassermann test. Vascular lesions were excluded by the gradual onset and the course of the illness, together with lack of evidence of circulatory abnormality. The blood pressure was within normal limits. Extrapyramidal lesions could not be excluded altogether, because of the generalized rigidity, paresis and abnormal movements; but there was no marked tremor at rest nor was the characteristic rhythm present. No history of any previous illness suggested post-encephalitic conditions. The patient was within the age limit for *paralysis agitans*, but his appearance did not suggest early stages of the characteristic signs. A slow-growing cerebral tumour, especially about the basal nuclei and internal capsule, was a possibility.

The gait, rigidity and so slight incoordination appeared to exclude cerebellar lesions.

Disseminated sclerosis appeared the most probable diagnosis, because of the mental symptoms accompanied by widely spread unsystematized and changing neurological signs, for example, the presence of extensor plantar response at one time with ankle clonus and its indefiniteness and apparent absence at another time; the greater rigidity of the right lower extremity than the left; the deviation of the tongue to the right; the altered speech; and the unusual movements, which subsided to a great extent. However, there was a lack of intention tremor, nystagmus and other eye signs.

Huntington's Chorea.

DR. C. HENRY showed a patient who was suffering from Huntington's chorea. The patient's father died aged ninety and his mother aged forty-six. Two brothers and one sister were alive and well, and no hereditary familial disease was known, but might exist, as the patient left home at too early an age to know anything of interest in this respect. He was a normal youth and was a ship's steward, voyaging to America from an early age. He was now forty-seven years of age, was married and had no children. He enlisted in 1915, suffered from a war neurosis

in France, and was discharged in 1919 with a pension for this. Ever since the war he had been very irritable and generalised irregular movements were noticeable in 1928.

His mental irritability appeared in 1920 and the motor irritability about eight years later, and authorities stated that in this disease the motor and mental factors were separately inheritable.

There were in this case no disturbances of sensation, the movements ceased during sleep, writing was difficult, and the patient progressed by standing on a very wide base, lifting his feet high in the air and bringing them down with a pronounced "stamp". If asked to stand still, he was motionless for three or four seconds, then there was a sudden flexion of the right arm, then of the left arm, then a sudden jerk forward of the head, followed by flexion of one leg at the hip, these movements being repeated in irregular order. His facial muscles contracted irregularly at the same time, this resulting in a very furrowed facies. His speech was quite intelligible, but somewhat "forced" and "staccato". All the deep reflexes were exaggerated. As a measure of desperation in this incurable disease he was given courses of tincture of stramonium, one cubic centimetre three times daily, since August, 1932, and his mental irritability was less pronounced, but his motor state remained unchanged or slightly worse.

Kyphosis.

Dr. Henry next showed a patient who was suffering from kyphosis, due probably to *spondylitis deformans*, but in which the bony changes shown in the skiagram did not seem sufficient to account for the curvature. The patient was forty years of age, married, motor driver, born in Victoria. He was the youngest of five and was a normal child and youth. He enlisted in the Australian Imperial Force in 1914 and served on Gallipoli and in France, and suffered from an aphonia, which was of doubtful causation, and from laryngitis. The latter, however, was not considered sufficiently severe to account for the continued aphonia. He was gassed in 1916 and returned to Australia in March, 1917, and was discharged unfit. In 1918 he reenlisted for canteen service and remained in Egypt until 1919.

In 1922 he was suspected of pulmonary tuberculosis, but in his sputum no tubercle bacilli could be found then or ever since. He first noticed his spine affected in 1918, before his second enlistment, when there was a slight curvature; and it had become steadily worse. He attempted suicide from the Harbour Bridge in October, 1932, and was admitted to the Repatriation Cottages as a voluntary patient.

The skiagram showed the site of the curvature to be in both the lumbar and thoracic portions of the spine, and there was only slight bony change in both these regions. Unfortunately the patient left before an anæsthetic was arranged to test whether the deformity was reducible under an anæsthetic. He complained of great pain on attempting to lie flat on his back in such a position as to straighten his spine. In view of the suspected hysterical nature of his aphonia, the question arose as to whether the kyphosis was partly due to functional and partly to organic causes.

Cerebral Syphilis.

Dr. Henry next demonstrated a case of concomitant strabismus together with epileptiform fugues occurring in a patient with cerebral syphilis. He was aged forty-four years, married, with one child; he was a commercial traveller and was born in New South Wales. He was a normal youth and was healthy up to his enlistment in the army in December, 1915. He was blown up in France in 1916 and says he has had pains in his head ever since. He returned from active service in 1918, when his eyesight and hearing were normal.

In 1924 he first noticed attacks of *petit mal* (he had five or six attacks in six hours). He said that images of things he had seen years before came before his eyes, the images were generally faces, and then he became momentarily unconscious. These had persisted up to the present. He left hospital on October 7, 1931, and between then and April 10, 1932, he experienced two fugues. He got out of bed in the night, walked 3·25 kilometres in his pyjamas and was found sitting in a motor car, with no recollection of his movements. On the second occasion he also walked about three kilometres. The left eye was normal, as were both fundi; the right eye showed a spasmodic strabismus, being directed towards the right. The field of vision showed concentric diminution and "everything seemed a long distance away". Acuity of vision was much disturbed for near vision, the patient being barely able to count fingers two feet away, but he could distinguish clearly objects a mile away. There was no diplopia and the fourth and sixth nerves were intact. There was no ptosis and no paralysis of the third nerve. The blood gave a positive reaction to the Wassermann test, but the cerebro-spinal fluid gave no reaction.

The case presents the question as to the relation between the fugues and the condition of cerebral syphilis.

Epilepsy.

Dr. Henry next showed a case of epilepsy in which the cause was stated by the patient's relatives to be injury by shell-shock while on active service. He was thirty-six, single, and was born in New South Wales. He was the second of six children and was a good scholar. His father died when he was thirteen and a half and he had to leave school and go to work; he improved his position by good work. He enlisted in July, 1915 and became a signaller in France; at Ypres a large shell exploded close to him and he was unconscious, but soon recovered and did not have to relinquish duty. From then on he became irritable, and a month later was gassed and sent to England; he arrived at Sydney in February, 1920.

He stated that he had his first fit just after his return, and had on an average two daily for a long time. Since admission to hospital in 1926 he had had fewer fits, about two a week, which were of the usual tonic and clonic nature, a feature being the long duration of the clonic state, which sometimes lasted up to half an hour according to the nursing staff. During the clonic state he roared and shouted continuously.

The interest in this case was the possibility of the explosion having caused the epilepsy. The patient's relatives stated that prior to that he had been of a somewhat superior mentality, which appeared credible, whereas now he was of the usual morose, irritable, retarded, selfish, epileptic character. A skiagram of his skull and brain revealed nothing amnormal.

Congenital Dementia Paralytica.

DR. J. A. H. MCGEORGE showed a patient who was suffering from congenital *dementia paralytica*. He was seventeen years of age and was an abattoirs employee. Twelve months ago his father noticed that he had become very irritable and querulous, finding fault with everything. He did not complain of any definite symptoms, but there was a change in his personality. For about five months prior to admission to hospital he was seen to be tremulous and at times his speech was almost unintelligible. The patient himself said that he felt remarkably well, except for "feelings like electricity" in his arms and legs. He believed that this was the result of being knocked down by a motor car about twelve months ago. He had been a healthy and apparently normal child prior to the onset of his present trouble. He was a full-time normal baby, weighing fourteen pounds at birth. He was born two years after the marriage and there had been no previous miscarriages. There were two younger children, both of whom were healthy. His mother had a miscarriage between the births of the second and youngest children. Both parents had always been healthy, except that both had had their appendices removed. The patient also had his appendix removed. The patient had been a good scholar and a bright, friendly child with normal interests.

On admission to Broughton Hall he was dull and apathetic, although expressing grandiose ideas of his ability. His speech was slurred and incoherent and he was irrelevant in conversation. His memory was unreliable and he was poorly orientated as to time and place.

Physically he was poorly developed, of dull appearance, with a slight degree of bilateral ptosis. There was a coarse "trombone" tremor of the tongue, and his pupils were inactive to light. He exhibited a tremulous condition of the extremities with voluntary movement, but there was no Rombergism. The knee jerks were exaggerated. Except for a systolic blood pressure of 160 millimetres of mercury and a diastolic pressure of 108 millimetres, other systems were clear. The Wassermann test gave a positive result with both blood and cerebro-spinal fluid. The cerebro-spinal fluid had a cell count of 63, gave positive globulin and Takata Ara reactions and a bicolour-guaiac reaction of 2444310000. His father and sister gave no reaction to the Wassermann test, but his mother gave a positive reaction.

He was inoculated with malaria and had several rigors, after which he became restless, confused, resistive, retaining urine, and later was apprehensive. He developed a furunculosis which responded satisfactorily to treatment. After this he became impulsive and aggressive for about a month and then settled down into a cheerful, expansive, euphoric condition in which he did some work in the grounds. His speech became more intelligible and at the time of his discharge he was quite amenable and a great deal more alert.

Manic-Depressive Psychosis.

DR. J. A. L. WALLACE showed two patients who were suffering from manic-depressive psychosis.

H.McD.C., aged thirty-six years, was admitted to hospital on December 1, 1924. He was married and a native of Scotland. The only point of note in the family history was that one sister was inclined to be hysterical.

The patient had convulsions as a child, was not a good scholar, became a messenger, then a bread-carter, and came to Australia in 1912. He enlisted in the army in 1916 and lost the left leg as the result of a gunshot wound. He was a Lewis gunner and worked up from Number 6 to Number 1. He was in France, also in Egypt; he was in hospital in England and was repatriated in 1919. He was an out-patient at the Randwick Hospital. He learned the wool trade and married in 1920. He became gate-keeper at a bank. At this time he was under the delusion that money had been stolen from the bank and that he was accused of the theft. He did not know of any money having been stolen, but "voices" accused him at night and he could not sleep. He went to the Prince of Wales Hospital, Randwick, and while there cut his throat superficially. His blood gave no reaction to the Wassermann test. He gave a history of moderate potus.

On admission to hospital he was well orientated and realized his condition. He was considered to be suffering from an unusual form of manic-depressive insanity. On admission his condition was diagnosed as such, although he had many characteristics of a typical schizophrenic. He was vividly hallucinated, believing that "voices" ordered him to commit suicide. He remained acutely depressed until 1925, when it became necessary to feed him by a tube daily and he resistively and deliberately vomited the food. He remained depressed for a further two years, when he became stuporose and would not speak or move. Late in 1930 he became talkative and said he was a Scotsman "because his feet stink and he loves Jesus". This was evidently the beginning of a manic phase, the depression having lasted for six years. At first his conversation was mostly obcene, but now he conversed in an amiable and witty fashion and had now been in a more or less manic state for two years.

F.S.W., aged forty-two years on admission to hospital on August 2, 1927, was married and a native of England. No family history was available. The patient was originally admitted through Number 13 Australian Auxiliary Hospital to Broughton Hall on December 4, 1918. He enlisted in November, 1915, at Victoria Barracks, Sydney, was sent to Egypt (Heliopolis) and returned to Australia in October, 1916. He had been employed in a printing office prior to enlistment and was reinstated after his return from the war. He was brought to Number 13 Australian Auxiliary Hospital by a Mr. G. from Scotland, who stated that the patient was a discharged man, aged thirty-five years, and who was "beginning to wander". There was no history of alcoholism. He was engaged to be married. On the Monday previous to admission he did not get up out of bed as usual in the morning and began to talk foolishly.

He was admitted to hospital in a state of acute mania, with much confusion; he was restless and noisy. The attack rapidly subsided and he was discharged recovered from the state of excitement after three months' residence. The following notes indicate the varied nature of the attacks.

In 1918 he was noisy, restless and irresponsible, shouting and singing; he recovered in four months.

In 1921 he suffered from acute mania, which subsided rapidly.

In 1922 he was excited and restless; he said he was the superior officer and must be obeyed; he recovered in three weeks and was discharged.

In 1923 he was noisy, restless and incoherent and rambled; he recovered and was discharged from hospital in two months.

In 1924 he was acutely maniacal; his condition settled down in five months and he was discharged.

In 1925 he was maniacal for eight months.

In 1926 he was maniacal for ten months.

In 1927 he was maniacal. He was well for short periods, but stayed in hospital. He was restless and loquacious and quite uncontrollable at times.

In 1931 he was noisy and maniacal; he dressed himself up in bizarre style.

Owing to the frequency of the attacks he had eventually to give up his work in the printing office.

It was pointed out that the patient had been latterly recovering from a severe attack of excitement. The attacks were of longer duration, and between the attacks there was more mental deterioration and inability to concentrate on any occupation; there was also dulness and general indifference to his environment. It was probable that he would eventually become more or less demented.

Schizophrenia and Fröhlich's Syndrome.

DR. J. A. L. WALLACE showed F.H.W.McC., aged eighteen, who on admission to hospital on July 6, 1919, was single. The family history had nothing of note. The patient was always strong prior to his enlistment in the army on June 6, 1916, at Lismore; he was in camp at Enoggera and at Salisbury Plains and Codford, England. The patient was not in action, but was returned as mentally deficient; he was not sent over to France; he suffered in England from cold and had "bad feet". He said he worried very much about his sister having "incurable dropsy" in Lewisham Hospital.

On admission he was slow and dull, he complained of neuralgia and that all his teeth ached and made his head ache. Mentally he was dull and slow and was also depraved in habits. Physically he was anæmic; his blood pressure was below normal. He was transferred to the list of certified patients at Callan Park on July 6, 1919. He had been an irresponsible patient, idle and apathetic.

His present condition was that he was very obviously suffering from Fröhlich's syndrome, that is, extreme hypopituitarism or *dystrophia adiposo-genitalis*. It was explained that this condition might be present before puberty, in which case the marked signs were a pseudo-feminine appearance—the broad pelvis and small hands and feet, smooth soft skin, small thin nails, with an absence of crescents at the base, were typical; the breasts were large and the genitals poorly developed, the pulse was slow, and epilepsy was common. In the present case the condition developed after puberty and the cause was probably degeneration and atrophy of the pituitary gland rather than neoplasm. In the skiagram the pituitary did not appear enlarged. There was lassitude and drowsiness, genital atrophy, and pseudo-feminine appearance. No alteration was seen in the fundus on ophthalmic examination, and the Wassermann test gave no reaction. The systolic blood pressure was 135 and the diastolic 85 millimetres of mercury. The hands were not small, one crescent only was present on the fingers. Pulse rate was 80. Urine was normal.

The patient's weight on admission was 52·1 kilograms (eight stone six pounds); it began to increase in 1928; he gained 31·5 kilograms (five stone) in two years, after which he continued to gain and was now 110 kilograms (seventeen stone six pounds) naked.

Schizophrenia and Obesity.

Dr. Wallace also showed J.A.M., aged twenty-five. On admission to hospital on August 18, 1924, the patient was single; he was born in England. His mother was of a somewhat emotional, loquacious type, and was of vigorous mentality. The father was a returned soldier, invalided with cardiac disease. The patient was a good scholar; he evidently tended to be reticent and never had many friends. In his military history there was a note that he was gassed during his active service and that he returned in 1919 and was employed in the Railway Department on electrical signal work. He failed at this; it was evidently beyond his capacity, and he became sleepless and worried.

He was admitted as a certified patient on August 18, 1924, and it was noted on his admission papers that he was suffering from arterial thickening, increased reflexes, unequal pupils and anæmia, that he gave no reaction to the Wassermann test, and there was a query as to his having a post-encephalitic psychosis. Owing to the anxiety neurosis from which his mother suffered in reference to this patient, every possible examination had been carried out on his various systems.

The patient, after years of irresponsible conduct, had settled down into a quiet, foolish, irresponsible state of dementia, with excessive development of bodily fat. He was still liable to be impulsive, and occasionally suddenly struck out at other patients. A sample of his writing showed the characteristics of a certain type of schizophrenia and was an example of wasted and misdirected energy.

The prognosis in this case was hopeless for recovery, but the patient might remain in a similar condition for several years.

Schizophrenia.

Dr. Wallace also showed F.G., aged twenty-four, who was suffering from schizophrenia. On admission on July 6, 1919, he was single. He was a native of New South Wales. The patient was the youngest of a family of six; the father was ten years older than the mother.

This patient was a very smart boy and clever at music. Six months before his enlistment he was in a clerical position at Orange; he resigned and came to Sydney, where he worked in a boot shop, and from there he enlisted. He was restless and thought he would like to get away and would enjoy the trip. He was always very steady and good living, but evidently had no attraction for the opposite sex. His sister often remarked that although he was young and bright, he was not engaged to be married when he enlisted. He wrote to his relatives regularly until he got ill. He informed them, when he returned, that he seemed to faint when he first became ill (at that time in Anzac Gully) and that he had also similar illnesses in France. Before he got ill he wrote to his relatives to say his nerves seemed to be going and he could hardly write.

On admission to hospital he was dull, depressed, uncommunicative, idle, untidy and indifferent. He remained a very irresponsible patient, sometimes impulsive and quarrelsome, incoherent, destructive and untidy.

At present he was quite disorientated, rambled incoherently in speech, was unable to look after himself, and was at times aggressive and homicidal.

Copies of letters written by the patient in 1916 showed the mental condition at that time and some indication of the onset of the schizophrenic state. For example, in a letter from Ismailia (February 2, 1916), after referring to various items of interest to his relatives, he wrote:

> After our spell on the Peninsula I should have been able to write them a treat, but I was too nervous for anything . . . I am longing to see you all again and have a gay old time with you all, so far I have managed to throw off all complaints, with the exception that I have many grey hairs—if that can be termed a complaint. Well I find myself guilty of taking so very little interest in things in general that I find it hard to put together a letter of interest. I think it is a characteristic of one after he has served a time in the military. However, you will know I often think of you all and remember all in my prayers at night and trust you all do the same for me.

In another letter, written about the same time, he wrote:

> I sometimes feel very neglectful for not writing more regularly than I do, but I assure you that 'tis writing under great difficulties I am, and find myself taking very little interest in things generally that 'tis almost impossible to put a letter together, 'tis a very bad way to let oneself drift, but I think 'tis a characteristic after one has served a time in the military . . . By the way, how do you like my last photo I have had taken, I look well and fat, eh? Still, my hair's getting grey. I feel very old and my own master.

These extracts indicated the early onset of the volitional weakness, which later became dominant when he was in a more or less completely disorientated mental state.

Dr. Wallace also showed D.C.S., aged twenty-nine, who was suffering from schizophrenia. On admission to hospital on September 20, 1920, the patient was single. He was a native of New South Wales. His family history showed nothing abnormal; the father was alive and had a high administrative position in a government department.

The patient was the youngest of a family of four; he was a healthy child; nothing abnormal was noted in his childhood. He joined the Navy when eighteen, served three years, and had to leave on account of a broken ankle. He enlisted in 1916 and was in France and was wounded at Ypres; he was sent to England and was discharged unfit in 1918. When he returned to Australia he began to get shaky and had slight nervous symptoms, he had headaches and body aches, and then developed head noises, "something to do with wireless, something communicating with the brain, something affecting him, some sort of spiritual influence". He also noted this in "other fellows"; he also saw a "great light" come into the ward at Randwick. He was admitted to Number 28 Australian Auxiliary Hospital on September 15, 1920, and still remained on a voluntary basis as a repatriation patient in the Repatriation Section.

The patient's present condition was that he still retained hallucinations of various types, and his speech was very disconnected and rambling. He was not employable, except occasionally, and had no initiative or application. He was taken out regularly for a motor drive each week, and when asked about this he made the most extraordinary statements about what occurred during his motor trip. He had a gunshot wound on the left upper arm and had atrophy and paralysis of the arm due to the musculo-spiral nerve being severed.

Discussion.

Professor W. S. Dawson stated that in the case of Paget's disease he presumed the mental deterioration was quite independent of the bony condition. In the very interesting case for diagnosis there appeared to be a pyramidal lesion with mental deterioration. Disseminated sclerosis might account for the very widespread lesions. In regard to the case of epilepsy, the fits appeared to be of the ordinary character of generalized epilepsy, and he presumed it was a case of traumatic epilepsy.

Dr. Kenneth Smith, in discussing the cases, referred first to E.D.McG., suffering from "epilepsy". Experience at the Repatriation Department showed that individuals gave extraordinary stories of the happenings during war service and that repetitions did not always correspond. In the case in question the actual history was as follows. After slight concussion in October, 1917, and slight gassing in 1918 the patient was returned to Australia in early 1919, and in consequence of a statement that a fit had occurred in July, 1919, he was placed in hospital for observation. This gave negative results. In December, 1920, following the receipt of a medical certificate, observation in "Russell Lea" was thought necessary. A report dated January 31, 1921, read:

Since admission he has had one seizure when apparently asleep. The sister states that he was breathing heavily and making moaning cries at intervals, some frothy saliva in mouth, face flushed, pulse good volume but slow, reflexes present. He has had another somewhat similar seizure and further observation is required.

On discharge from hospital on March 7, 1921, the report read:

Since January 31, 1921, patient has had one fit of a functional nature.

In November, 1921, further hospital observation was instituted and after two months a definite diagnosis of epilepsy was made and accepted as a war disability. The general policy of the Repatriation Commission was to accept as pensionable any disability developing during war service abroad, and especially if there were a definite record of field service. The generosity of this policy was exemplified in the case of Fröhlich's syndrome, which had been demonstrated, where the individual concerned got no further than England.

In reference to the case of kyphosis Dr. Smith said he would like to hear Dr. Royle's opinion, as the bony changes seen in the film offered no explanation of the man's symptoms and condition. The general physical state and mentality offered a better explanation.

Dr. Smith was in agreement with Professor Dawson that the Paget's disease was only an accidental association with the mental state. There were several cases of Paget's disease in the departmental records where there was no mental alienation.

The case of Huntington's chorea was particularly interesting, as this diagnosis had been made in some four cases where there was no hereditary history. After all, the disease must make a beginning somewhere in the history of a family, and there was no reason to assume that cases could not arise *de novo*. In regard to the syphilitic case, despite statements made by the man, enlistment occurred in December, 1915, and there was no history of previous service. The disability in this case had not been accepted as war caused. He did not propose to discuss the reasons for this decision, but reiterated that it was very necessary to have chapter and verse for all the facts put forward to the Repatriation Department. The schizophrenic patient (J.A.M.) had changed in a remarkable manner since Dr. Smith originally saw this patient. He was tall and gaunt, with sharp features, but during the last six months the whole appearance had altered. He was now well nourished and fat, with entirely different features, and it would be difficult to credit the change unless one had seen the patient previously.

Dr. Rowland Edwards said he could give some relevant particulars about the patient diagnosed as having cerebral syphilis. The man belonged to a well known family of markedly extrovert type. One member of the family had long been known as "queer" to his associates. The patient had brothers and sisters more intelligent than ordinary. But the neurotic strain in the family was shown by the mother being hysterical (she was also asthmatic). Her attacks had taken various forms. The last one of which Dr. Edwards had knowledge was a sudden loss of memory on learning that her daughter was to be sent by a government department, in whose service she was, a considerable distance into the country. In view of the history and the family disposition, it seemed to Dr. Edwards that the fugues might not be due to syphilis, but might be an expression of that type of neurosis so often found in neurotic extraverts, namely, hysteria.

Dr. A. W. Campbell said that they had seen a number of interesting cases and he had to thank the demonstrators not only for presenting them instructively, but for providing a typewritten history with each patient. He had little to add to the discussion. He would only say that he had occasionally seen examples of Paget's disease outside mental hospitals. One patient was a woman whom he had been able to observe for years, and it was interesting that as an early sign she had found that her false teeth would not properly fit her gums, next the bridge of her nose broadened disfiguringly, and lastly she had not been able to get a hat to fit her head. In regard to the case for diagnosis, he said that when to a neurological problem mental disorder was added, the diagnosis became doubly difficult because the patient was unable to give any facts. In this case, however, the dominant feature was the rigidity of the arms and legs. It was not quite that of decerebrate rigidity, but there might be partial decerebrate rigidity, provided the lesion lay at the appropriate level in the brain stem, at the level of the anterior *corpora quadrigemina*. Possibly the patient had a lesion in that position.

Dr. Henry, in reply, thanked Professor Dawson for his suggestion that the epilepsy in the case shown was of traumatic origin, and it seemed probable that it was so. The interesting family history in the case of cerebral syphilis given by Dr. Edwards suggested that the fugues were possibly hysterical and not associated with the luetic condition.

Dr. Rainbow stated that there was no intracranial pressure in the case shown, but recently another patient with Paget's disease, also insane, had been admitted to Callan Park. When he came he was in a most inaccessible state, and although delusional, could understand what was said. At *post mortem* examination the right side of the skull was rather more prominent, but it was found that the thickness of the skull was uniform throughout, although it seemed to have a thickening owing to the external prominence.

Dr. Wallace stated that the cases he had brought forward, of the manic-depressive and schizophrenic type, were commonplace and everyday cases in the mental hospitals department, but he considered they might interest the meeting as they were all accepted as attributable to war service.

NOMINATIONS AND ELECTIONS.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Corlis, Wilson Leighton, M.B., 1931 (Univ. Sydney), Coast Hospital, Little Bay.

King, Callaghan John McCarthy, M.B., Ch.M., 1923 (Univ. Sydney), c.o. Dr. W. H. Cook, Young.

McKinnon, Malcolm Charles, M.B., B.S., 1929 (Univ. Sydney), Albion Park.

Macleod, Daniel, M.B., Ch.B., 1929 (Edinburgh), Lenakel, Tanna, New Hebrides.

Obituary.

MICHAEL HENRY DOWNEY.

We regret to announce the death of Dr. Michael Henry Downey, which occurred on April 17, 1933, at Adelaide, South Australia.

JOHN WILLIAM SPRINGTHORPE.

We regret to announce the death of Dr. John William Springthorpe, which occurred on April 23, 1933, at Melbourne, Victoria.

Proceedings of the Australian Medical Boards.

TASMANIA.

The undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Davis, John Reginald, M.B., B.S., 1908 (Univ. Melbourne), Westbury.

Clarke, Edmond, M.B., B.S., 1932 (Univ. Melbourne), Launceston Public Hospital, Launceston.
Kingsmill, Caroline Mary, M.R.C.S., L.R.C.P., M.B., B.S., 1927 (London), Glenorchy.
May, Alexander Joseph, M.B., M.S., 1924 (Univ. Melbourne), M.R.C.P. (London), Campbell Town.

Books Received.

CRITERIA FOR THE CLASSIFICATION AND DIAGNOSIS OF HEART DISEASE, by the Criteria Committee of the Heart Committee of the New York Tuberculosis and Health Association, Inc.; Third Edition; 1932. New York: New York Tuberculosis and Health Association. Demy 8vo., pp. 141, with illustrations.

THE DIABETIC LIFE: ITS CONTROL BY DIET AND INSULIN, by R. D. Lawrence, M.A., M.D., F.R.C.P.; Seventh Edition; 1933. London: J. and A. Churchill. Sydney: Angus and Robertson, Limited. Demy 8vo., pp. 226, with 10 illustrations. Price: 8s. 6d. net.

THE PRACTICAL MEDICINE SERIES OF YEAR BOOKS: GENERAL SURGERY; Series 1932; 1933. Chicago: The Year Book Publishers. Crown 8vo., pp. 816, with illustrations.

Diary for the Month.

May 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
May 2.—Western Australian Branch, B.M.A.: Council.
May 3.—Victorian Branch, B.M.A.: Branch.
May 4.—South Australian Branch, B.M.A.: Council.
May 5.—Queensland Branch, B.M.A.: Branch.
May 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
May 12.—Queensland Branch, B.M.A.: Council.
May 16.—New South Wales Branch, B.M.A.: Ethics Committee.
May 17.—Western Australian Branch, B.M.A.: Branch.
May 17.—Victorian Branch, B.M.A.: Clinical Meeting.
May 18.—New South Wales Branch, B.M.A.: Clinical Meeting.
May 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
May 24.—Victorian Branch, B.M.A.: Council.
May 25.—South Australian Branch, B.M.A.: Branch.
May 25.—New South Wales Branch, B.M.A.: Branch.
May 26.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. W. S. McGillivray (B.M.A.) has been appointed Deputy Commissioner of Public Health, Western Australia.

* * *

Dr. H. S. Covernton (B.M.A.) has been appointed Acting Responsible Medical Officer, Mareeba Babies' Hospital, South Australia.

* * *

Dr. F. M. G. Punch (B.M.A.) has been appointed Acting Medical Officer in Charge of the Lock Hospitals at the State Penitentiary and State Reformatory for Women, Long Bay, New South Wales.

* * *

Dr. C. W. England (B.M.A.) has been appointed Government Medical Officer at Dorrigo, New South Wales.

* * *

Mr. M. J. Holmes (B.M.A.) has been appointed Acting Director-General of Health and Acting Director of Quarantine for the Commonwealth.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", page xvi.

Children's Hospital (Incorporated), Perth, Western Australia: Junior Resident Medical Officers (male).
Launceston Public Hospital, Launceston, Tasmania: Resident Medical Officer (male).
Mater Misericordiæ Children's Hospital, Brisbane, Queensland: Resident Medical Officer.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
South Australian: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to "The Editor", The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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